THE
BRITISH JOURNAL OF SURGERY

VOL. XXXVI OCTOBER, 1948 No. 142

RADICAL MASTECTOMY

THE TECHNIQUE AND THE COMPLICATIONS

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INTRODUCTION

THE disadvantages of the radical operation are well known—*mutilation, post-operative swelling of the arm, and operative mortality and morbidity*. Of these, with care, it should be possible to eliminate almost entirely the last two, and measures to this end are discussed later under the appropriate headings. Post-operative swelling of the arm, while one does not wish to belittle this all too common complication, is fortunately but rarely seriously incapacitating. The disfigurement needs no comment except to say that it is common to the simple (*Fig.* 152) as well as the radical operation.

All these factors must weigh heavily with the conscientious surgeon. The removal of an eye or a limb is equally abhorrent to him yet may prolong a fruitful life for many years. So the radical operation for carcinoma of the breast must not be jettisoned just because it is distasteful until there is something better to replace it. Indeed, I sometimes think that we look upon this organ too much through the eyes of the male and perhaps attribute to it a sanctity and indispensability with which it is not always so regarded by members of the sex in which it is found in such varying forms (*Figs.* 153, 154).

Results.—The operation of radical mastectomy is not yet obsolete. In the treatment of the early case no other known method has yet produced results as satisfactory over a 10-year period. In the series of Stage I cases published by Gordon-Taylor (1938) and in the Leeds series (1926) of gland-free cases 84 per cent and 91 per cent respectively of the patients operated upon were alive ten years later. This figure approximates very closely to the chances of survival amongst the general population in the same age-periods and must therefore be regarded as very satisfactory. Until convincing evidence is available it is difficult to see how the thoughtful surgeon can abandon the operation of radical mastectomy for more conservative procedures in this group of patients.

The Operative Mortality.—There is evidence that the operative mortality can be very low. Cade (1948) has recently collected the figures of twenty-two British surgeons. There were 11,014 radical mastectomies with 182 deaths, a mortality of 1·65 per cent. Even this is an over-estimate of the present position, for it is based upon operations ranging over the last twenty years. In this series of 170 consecutive radical mastectomies there was no operative mortality—that is to say, no patient died within one month of operation or while still in hospital. While this desirable issue may in some measure no doubt be attributed to good fortune, it is to be regarded rather as a sign of the times and not as an exceptional experience. The modern operation, in good hands, with all its ancillary advantages in anæsthesia, penicillin, and the sulphonamide drugs, probably has a mortality of less than 1 per cent.

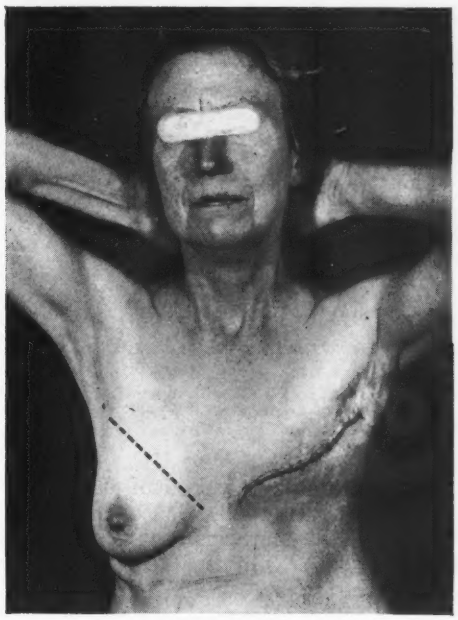


FIG. 152.—The incision used for simple mastectomy. It is made in the long axis of attachment of the breast, which is oblique and not vertical or horizontal.

Estimation of the over-all operative mortality throughout this country can only be a guess, but since the published operative fatality rates reflect the best, rather than the worst, results obtainable, it is probably not less than 2 per cent and may be as high as 5 per cent in inexperienced hands, if the somewhat extended definition of operative mortality used above is applied. Of the deaths that do occur, it is probably true to say that a minority might have followed any operation of equal magnitude, but that the majority occur because the patient is unsuitable

for or unprepared for the operation. It is towards the prevention of these deaths that surgeons can effectively turn their attention.

The Causes of Death following Operation.—The commonest causes of death have in the past been attributed to shock, to sepsis, and to pulmonary

FIG. 153.—St. Agatha, as patron saint of Fertility. A statuette in alabaster preserved in the catacombs beneath the Church of St. Agatha in Malta.

complications, including embolism—S. F. Marshall and J. Higginbotham (1938), S. Roden (1944), S. L. Joyce (1936), F. S. Mathews (1932), C. Braine and G. Massie (1926) (Guy's Hospital series), G. Gordon-Taylor (1938), W. H. G. Jessop (1936) (University College Hospital series), A. C. Perry (1925) (London Hospital series). While pulmonary embolism is still a serious post-operative problem, it should be possible to eliminate almost entirely the other three factors.

FACTORS INFLUENCING OPERATIVE MORTALITY AND MORBIDITY

INDIRECT FACTORS

Before considering the specific measures which may be taken towards reducing operative mortality and morbidity it may not be amiss to review some of the indirect factors which may be influencing surgeons in their attitude towards this operation and be responsible for some of their loss of interest and, possibly, loss of care.

First: The operation is a very old one so far as major surgical procedures go and was performed very much as it is to-day more than fifty years ago. It has, therefore, lost much of its original appeal, for it is no longer a pioneer achievement—indeed, it is in danger of being regarded as a somewhat hackneyed affair, for there has been little change in its execution over the years.

Second: The operation suffers in its simplicity to-day by comparison with the more gargantuan procedures which are there to distract the enthusiastic surgeon of this era as compared with his forebears of half a century ago, to whom a radical mastectomy was the most extensive surgical undertaking known. With this almost inevitable transfer of interest to other fields more romantic and exciting, there is the danger that the radical operation for cancer of the breast will be indifferently performed and that the elementary precautions which, if employed, render it such a safe operation may be but lazily applied.

Third: The growth of radiotherapy has had a lulling, soporific effect on some surgeons, who have

FIG. 154.—The patron saints, St. Lucy (the eyes) and St. Agatha (the breasts), carrying their respective offerings. (*By courtesy of the Wellcome Historical Museum.*)

been inclined to transfer their confidence too wholly and too prematurely to this powerful adjuvant. It may have made some surgeons less thorough in their operation, relying on post-operative irradiation to deal with living cancer cells they may have left behind.

For these various reasons the experienced surgeon too often places the operation at the end of his list, and the occasional surgeon, because of the infrequency of an operative fatality, too often regards the operation as within his compass.

GENERAL PREVENTIVE MEASURES

Apart from the usual general assessment of the patient's fitness to withstand a major operation, including an abdominal and pelvic examination to exclude palpable secondary deposits, two elementary investigations are of vital importance, namely :—

1. Radiography of the chest, spine, and pelvis.
2. The estimation of the hæmoglobin level in the blood.

1. **Radiography.**—Radiographs of the *chest*, *spine*, and *pelvis* to exclude the presence of metastases should be taken pre-operatively as a routine. It is only in this way that a silent *pleural effusion*

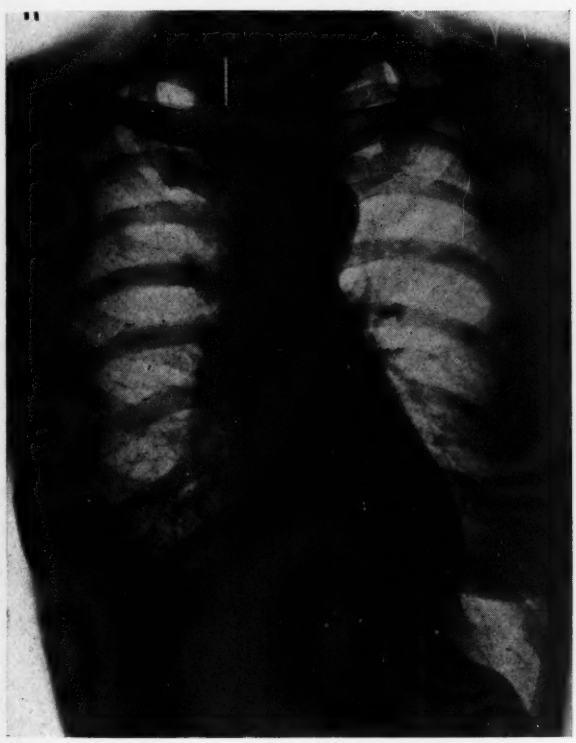


FIG. 155.—Preliminary radiograph of chest showing symptomless pleural effusion (right).

FIG. 156.—Preliminary radiograph of chest showing metastases in the left lung.

(*Fig.* 155) or a *symptomless secondary deposit in a lung* can be discovered (*Fig.* 156). There is little doubt that from time to time patients are submitted to the radical operation with metastatic deposits already present in the skeleton or more commonly in the chest. Operation in these circumstances can only accelerate the patient's end and may explain some of the early post-operative fatalities under anæsthesia that have been reported.

2. **Hæmoglobin Estimation.**—If the element of chance in radical mastectomy (or any other major operation) is to be cut down to the minimum, the routine estimation of the pre-operative level of hæmoglobin is fundamental. Just occasionally a *very low level indeed* will be found on admission (*Fig.* 157). To operate on a patient whose hæmoglobin is, for example, only 60 per cent is to take a quite unnecessary risk, particularly if we bear in mind that a further substantial reduction is likely as the result of the operation.

There is no question of advising a blood transfusion for every one with cancer of the breast. The majority will not require blood at all. It is, however, important to ensure that patients should have a hæmoglobin at a reasonable level in the immediate post-operative phase, if we want to place them under the most favourable conditions for recovery.

The procedure advised is as follows :—

Pre-operative Hæmoglobin less than 70 per cent —Pre-operative Transfusion : Patients who on admission have a hæmoglobin level in the 60's or 50's will require a blood transfusion as a pre-operative measure : the dosage should be calculated so as to raise the hæmoglobin level to 80 per cent.

Pre-operative Hæmoglobin more than 70 per cent but less than 80 per cent—Transfusion during Operation : It will be wise to give most of these patients 1 pint of blood during the operation. This will apply particularly to the frail and to the women over 60. In the younger, physically stronger age-groups it may be safe to withhold blood transfusion.

The Fall in Hæmoglobin produced by Radical Mastectomy.—To discover the fall in hæmoglobin produced by radical mastectomy the hæmoglobin levels were estimated on the day before operation and on the fourth post-operative day. In 55 patients in whom this investigation was made, it was found that the average drop produced by the operation was 13·5 per cent. Patients given a blood transfusion during the operation were excluded from this investigation. Although there was a drop of only 10 per cent or less in 22 cases, there was a drop of 20 per cent or more in 14 cases of the 55. This figure seems all the more significant in view

of the fact that one of the features of the operation here described is that it has been designed to reduce blood-loss to a minimum. It is possible, therefore, that in some methods the average blood-loss is considerably higher than in this series.

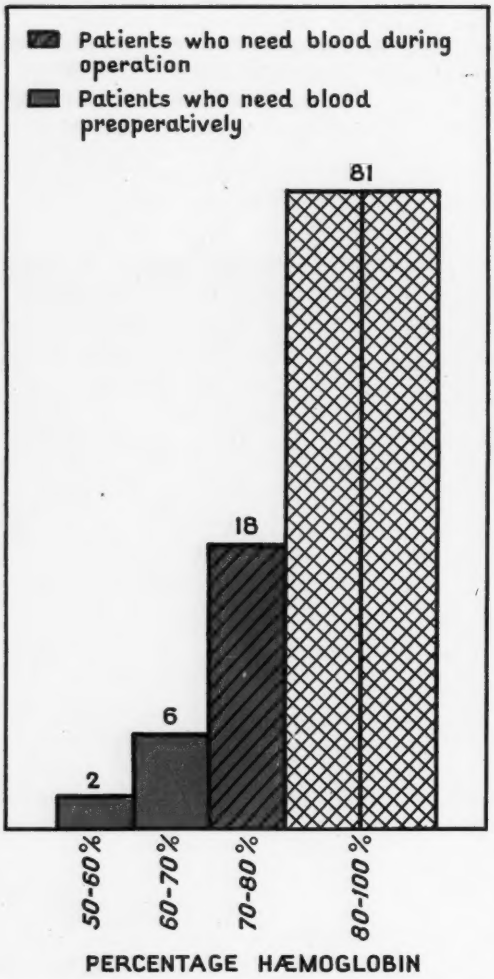


FIG. 157.—Hæmoglobin levels (on admission) in 107 cases of carcinoma of the breast.

Measures against Shock.—Gentleness and hæmostasis: nothing exceeds and nothing equals their importance. Too often indeed is this operation little better than an "amputation of the breast"—horrible phrase, with all it implies of speed, hæmorrhage, and rough handling (*Fig.* 158). With proper care the loss of a patient from shock following radical mastectomy should never happen. There are, however, so many factors that can conspire to this end that active measures must be taken to prevent it developing. Anti-shock measures are aided: by avoiding exposure to cold; by abandoning the use of wet packs; by giving fluid intravenously during the operation to 'poor-risk' cases; by keeping blood-loss to the minimum; by completing the operation in a reasonable time; by avoiding tension in the approximation of the skin-flaps; by avoiding tight bandaging across the chest; and by lifting and transporting the patient gently.

Measures against Infection.—Sepsis is still an occasional cause of an operative fatality and a frequent cause of operative morbidity. The liability to sepsis can be reduced to a minimum by the following preventive measures:—

1. A thorough pre-operative *cleansing* with soap and water of the whole chest area, particularly the skin of the *flank* and of the *submammary sulcus* in *stout women.*

2. Adopting a *towelling* technique which ensures the sterility of the vulnerable area on the flank.

3. Avoiding *damage to the blood-supply* of the lateral flap (*see* Flap Necrosis).

4. Using the *diathermy* in such a way as to avoid massive tissue necrosis.

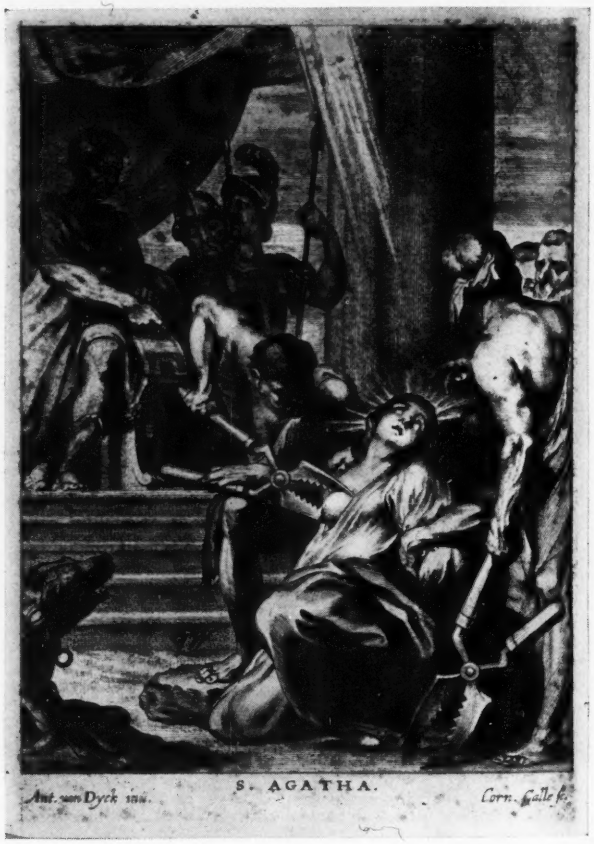


FIG. 158.—The martyrdom of St. Agatha.

5. Diminishing the opportunities for *cross-infection* from one part of the wound to another—first, by sealing off the drainage opening from the rest of the wound; and, second, by avoiding exposure of the clean and the potentially dirty part of the wound at the same dressing.

6. Delaying deliberate *active movement* of the arm on the affected side until the wound is healed, or separation of the skin edges and serum collection may occur.

Respiratory Complications.—These are related in the operation to much the same causes as in other operations, but careful selection of cases and avoidance of intubation trauma are important considerations.

Intravenous Fluid.—Many surgeons consider that it is unnecessary to give fluid intravenously

during the operation. A generalization of this kind is as unwise as is that of the opposite school who would give fluid intravenously as a routine. We try to discriminate between our patients; and we find that while fluid can safely be withheld from many of those under the age of 45, there are few over this age who do not appear to benefit from it. It is our practice to give all poor-risk cases 1 pint of plasma by needle into the saphenous vein at the ankle during the operation. The fluid is introduced at a fast drip rate so that it has all run in before the patient is returned to the ward. Blood is only substituted if the pre-operative hæmoglobin is below 80 per cent. We have so far had no cause to regret this action, and the ward sisters tell us that they are less frequently troubled by post-operative shock than when fluid is omitted. There is certainly no doubt that if fluid is to be introduced it is much more effective in its prevention of shock if given during the operation than afterwards.

BIOPSY

The clinical diagnosis was uncertain in 54 instances in this series of 170 cases. One would like to see more uncertain cases. My procedure has been to make an incision through the skin overlying the tumour and to deepen the incision so as to cut directly into the centre of the tumour in situ. I prefer not to excise the growth to examine it, as this involves opening up large veins on the periphery of the tumour by way of which metastases may be disseminated even if diathermy is used for the excision. The centre of a hard growth is almost avascular so that the chances of dissemination are reduced to a minimum by this method. If the operator is undecided after cutting into the growth he must excise it with the diathermy and either hand it over for immediate frozen section or close the wound and send the specimen to the laboratory. My own experience has been that it is in just those cases in which the surgeon is still uncertain after cutting into the tumour, that the pathologist also is going to have most difficulty in making up his mind. For this reason I do not use the frozen section technique, although in the very experienced hands of a few it is said to be reliable. In these circumstances I believe it is wiser to allow the pathologist the extra time necessary to dehydrate the specimen thoroughly, with the advantages in staining and examination which such preparation makes possible. While a second anæsthetic may be necessary the delay occasioned by this more cautious method is not great. An unhurried examination can be carried out and a reliable report obtained within twenty-four hours of sending the specimen to the laboratory.

THE OPERATION OF RADICAL MASTECTOMY

Equipment.—

Amputation Knife.—No special instruments are required, but there is one which should be abandoned for all time: this is the *amputation knife* which was originally, and still is occasionally, used for raising the flaps by a process of transfixion—a crude and bloody method which has nothing to commend it. It is advisable if a knife with detachable blade is used, to have a number of spare blades available, as there is no greater aid to a clean and even dissection than a knife with a razor-sharp edge.

Warmth.—To protect the flaps and to keep the exposed areas warm it is an advantage to use *dry* packs, hot from the autoclave. I have found them an improvement upon *hot wet* packs which so soon become *cold wet* packs and chill the patient. Wet packs are best reserved for hæmostatic purposes at the end of the operation and only applied to the chest wall after the breast has been removed.

Swabs.—It is a great convenience to be able to use rather larger swabs than normal. The size suggested is 5 in. × 5 in., with a thin layer of wool incorporated between the layers.

Diathermy.—The diathermy should never be used for the skin incision or in the axilla. It should not, in my opinion, be used to coagulate the main perforating arteries or the larger tributaries of the axillary vein. It has to be used with particular caution in *thin* subjects and in the region of the *base of the flap.* As a method of raising the flaps I think its disadvantages outweigh its advantages, particularly in inexperienced hands, but I admit to being prejudiced. I believe it is the commonest cause of flap necrosis. The heat generated at the base of a flap raised by diathermy has to be felt to be appreciated and it undoubtedly on occasion is responsible for interfering with the blood-supply. I prefer to reserve its use for the coagulation of the smaller blood-vessels only. If carelessly used for coagulation, it can cause widespread tissue necrosis, which in turn produces an exudation of serum and an increased liability to sepsis. Care should be taken to see that the neutral electrode pad is placed over healthy tissues, for the slight heat developed in the pad has been known to light up an old infective focus. The loin is safer in most cases than the lower limbs, which may have varicose tendencies.

Preservation of Non-essential Structures.— While all operators must find it repugnant to have to remove the female breast, there are some who, having taken the decision, are still loth to remove the pectoral muscles or to sacrifice the nerve to latissimus dorsi even when this would make possible a more thorough clearance of the axilla.

The Pectoral Muscles and Functional Disability.—The two pectoral muscles must be removed. It is not the loss of these two muscles which makes radical mastectomy a mutilating operation, but the loss of the breast itself. Their removal is required in order to provide adequate exposure in any operation claiming to clear the axilla of malignant deposits. Neither the apex nor any other part of the axilla can be properly dissected so long as these muscles remain in situ. A partial clearance has no place in the cancer surgery of this area. The occasional practice of retaining the pectoralis minor is to be condemned. Functionally, it is an unimportant little muscle and its presence obscures the most vital part of the area to be dissected. *Functional disability* following radical mastectomy should be rare. It is most commonly due to *skin loss,* less commonly to a badly placed incision. In my experience it is not related to loss of the pectoral muscles, or failure to employ early movements of the arm post-operatively. It is indeed surprising how slight

is the effect of the removal of the two pectorals upon the movements of the shoulder and the stability of the shoulder girdle. Three months after the operation there is remarkable power in the arm of the affected side. The ability to raise the arm vertically is the most delicate measure of residual functional disability (*Fig.* 159).

The Intercostal-humeral Nerve.—The intercosto-humeral nerve and its counterpart in the first space, the lateral cutaneous branch of the first intercostal, exert a strange fascination upon some operators, who will take great pains to preserve these structures

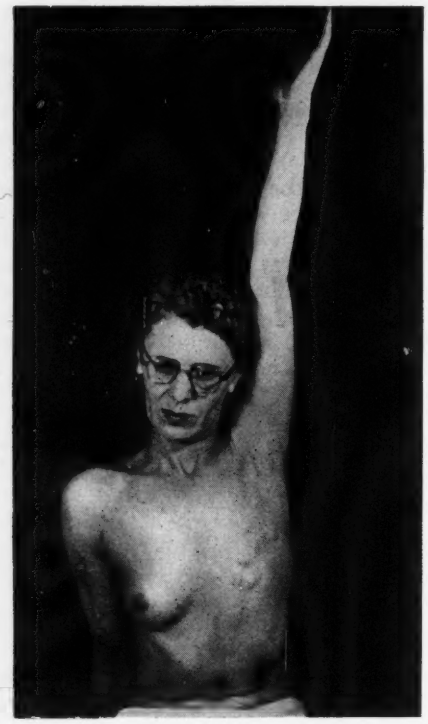


FIG. 159.—The measure of residual functional disability following radical mastectomy.

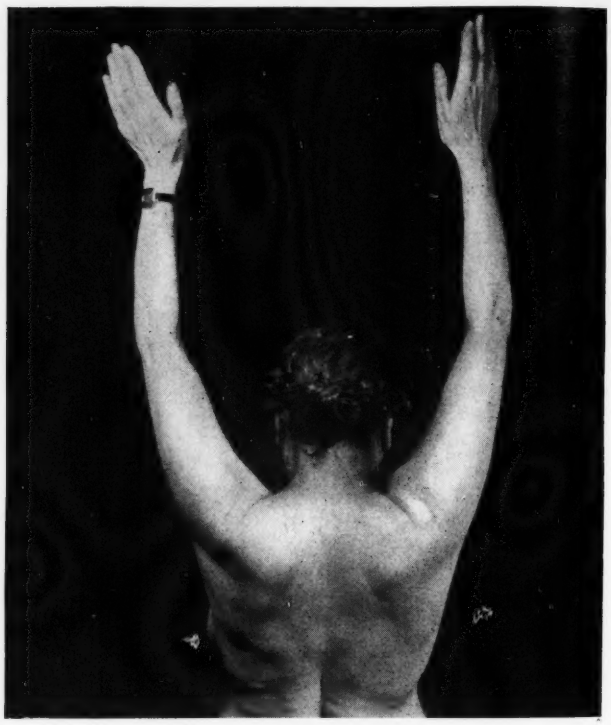


FIG. 160.—The nerve-supply to latissimus dorsi has been divided on the right side without any impairment of movement at the shoulder.

and their intercommunicating nerve-plexus, at the expense sometimes of having their attention diverted from malignantly infiltrated tissues nearby.

The Nerve to Latissimus Dorsi (Middle Subscapular or Thoracodorsal Nerve).—There should be no hesitation in sacrificing the nerve to latissimus dorsi if there is any question of its impeding the thoroughness of the block dissection of the axilla. It has to be remembered that this is an operation designed to eradicate a widespread malignant process and that while wanton destruction is pointless, half measures are worse than useless. There are occasions when there appear to be no enlarged lymph-nodes and no visibly malignant tissues and, in these circumstances, the nerve to latissimus dorsi should be isolated and left as deliberately as it should be removed in more advanced cases. I find that I divide it more often than I leave it. In a series of cases examined by Dr. Clive Shields in the Department of Physical Medicine at St. George's Hospital, little or no disability has been noticed in these patients as compared with control cases and with the opposite side. In spite of an absolute R.D. being present in the latissimus dorsi, as shown by a failure to respond to galvanic and faradic stimulation, none of the patients examined by us complained of any limitation of movement of the arm on the affected side as a result of the operation (*Fig.* 160).

The Clavicular Head of Pectoralis Major.—There is no advantage to be gained from sacrificing as a routine the clavicular head of the greater pectoral. The malignant process rarely spreads to the outer side of the neurovascular bundle and recurrence in this situation or in the clavicular portion of the muscle must be exceedingly rare—if it ever happens. Some critics have questioned the advantage of leaving the clavicular head on the grounds that it becomes a non-functioning remnant as a result of the destruction of its nerve-supply during the operation. This has not been our experience. The nerve-supply is not destroyed. In a series of patients examined, voluntary movement was present in the clavicular head in all cases, and electrical tests produced normal reactions and normal electronic pulsometer curves (*Fig.* 161).

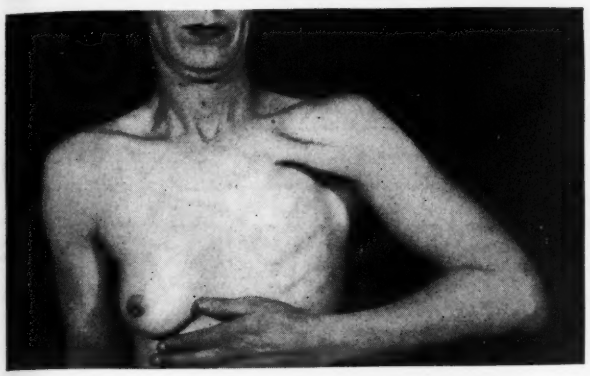
The Cephalic Vein.—Similarly the cephalic vein should be carefully preserved, and this will be automatic if the clavicular head of pectoralis major is undisturbed. Deliberate division of, or accidental damage to, the cephalic vein at the beginning of the operation may be regretted if subsequently the main axillary vein has to be resected or tied for any reason such as injury or encirclement by growth.

The Requirements of a Radical Operation.—*The Skin.*—

The area of skin to be excised.—Removal of a margin of skin well clear of the edge of the growth

is essential—irrespective of the use of radiotherapy—or a crop of secondary nodules will appear in the region of the scar within a few months of the operation (*Fig.* 162).

The area of skin available for the flaps.—The area of skin available for fashioning the flaps must be worked out *before* the operation. This assessment is an important preliminary and aid in determining whether the initial method of treatment of the case shall be *surgical or radiotherapeutic.* In making this assessment it will be found that patients



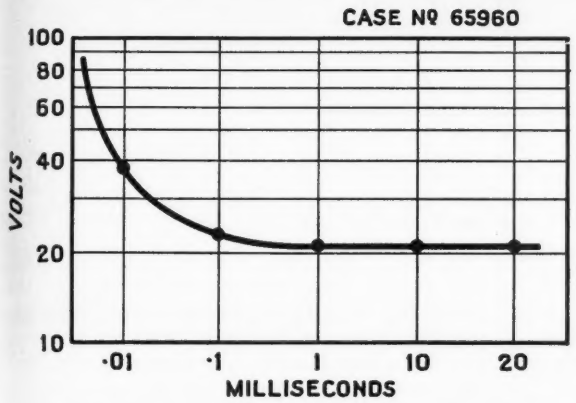


FIG. 161.—Photograph showing voluntary movement persisting in the clavicular head of pectoralis major after mastectomy. The graph shows the normal curve for the remaining portion of the left pectoralis major.

fall into two categories—a small number in whom a primary removal of the breast would leave a defect between the skin edges, and a larger number in whom it will be possible to approximate the skin edges without tension in the usual way. *Pre-operative* X-ray therapy is of particular advantage and has a special application in the treatment of the smaller group—that is, in those patients in whom it is judged that, because of a *shortage of skin*, primary removal of the breast (and tumour) would leave a gap between the edges of the skin-flaps. A shortage of skin, although not invariably present, is commonly found in both stages I and 2—(i) when the tumour is large or even more often when the *breast is unusually small*, as, for example, in males; (ii) when the tumour is attached to the skin over a wide area; and (iii) when the tumour has ulcerated through the skin.

A *defect* left at the end of the operation between the edges of the flaps has two disadvantages: *First*, it can only heal by skin-graft, or by granulations and scar-tissue, which form a patch vulnerable to subsequent post-operative therapy; and *secondly*, and more serious, healing is often protracted so that the start of post-operative therapy is delayed sometimes to a point when its usefulness is open to question.

FIG. 162.—Secondary skin nodules resulting from too restricted an excision of skin (charted for record purposes).

If, by careful clinical assessment, *we can anticipate* such a defect arising—and to do that the area of skin available for the flaps must be worked out beforehand and not for the first time on the operating table—then we should seriously consider giving the X-ray treatment as a pre-operative measure (Riddell, 1948).

FIG. 163.—Post-mastectomy defect (skin grafted) in a patient who has already received pre-operative X-ray treatment.

It goes without saying that pre-operative irradiation does not in any way absolve the operator from still adhering to the principle of excising a wide area of skin clear of the growth. The point is that once the course of therapy has been given, delay in healing of the wound has not the same significance, as it holds up nothing except the ordinary convalescence of the patient (*Fig.* 163).

'Block Dissection' of the Axilla.—Cancer surgery, if it is to be effective, must be radical. Above all is this true of the early case. Incomplete procedures may have their place in the treatment of the advanced growth, but in the early case they can, in the present state of our knowledge, be neither justified nor pardoned. In the past, while emphasis quite rightly has been placed on the importance of removing adequate skin, too little has been placed on the still greater importance of a *radical block dissection* of the axilla. It is upon the absolute thoroughness of this step that radical mastectomy as an operation eventually stands or falls.

Hæmostasis.—The prevention of excessive hæmorrhage should be deliberately planned as one of the major objectives of the operation. Blood-loss is reduced :—

1. During the elevation of the lateral flap by controlling bleeding points by swab pressure rather than by the use of artery forceps.

2. By delaying the elevation of the medial flap until its blood-supply has been controlled.

Deep Fascia : Extent of Removal.—Removal of the 'pectoral' fascia alone is not sufficient. The *deep* fascia over the upper part of the rectus abdominis, the external oblique, and the serratus anterior must also be excised. The limits are :—

Above—to the clavicle.
Medially—to the middle line.
Laterally—to the edge of latissimus dorsi.
Below—the long axis of the 8th rib.

TECHNIQUE

The operation here described does not introduce any new concept in the surgery of cancer of the breast, or violate any of the canons of pathology upon which the original operation was founded and upon which it has stood the test of time. This is indeed a tribute to its distinguished originators, William Stewart Halsted (1895) of Baltimore and Mitchell Banks (1883) of Liverpool.

The old operation, however, was something of a smash-and-grab undertaking, and hence was well-named 'amputation of the breast'. Modern anæsthesia and anti-shock measures have made it possible to carry out the same operation planned with rather more regard for anatomical details and natural planes of cleavage than was feasible when hæmorrhage was an obscuring factor and speed an all-important consideration.

The account which follows differs only in points of detail from other descriptions of the operation and claims no special distinction or superiority to the exclusion of other methods. The main emphasis is to be placed rather on the thoroughness of the procedure than on any special variation in the steps concerned with achieving this and upon the importance to be attached to keeping blood-loss to a minimum.

Position of the Theatre Nurse.—The position of the nurse taking the case is important. She should stand at the *head of the table.* From this position she gets an uninterrupted view of the operative field and so can serve the surgeon much more effectively than if she is standing at the side or foot of the table (*Fig.* 164).

Position of the Arm.—A deltoid palsy is still an occasional complication of this operation. It arises as a result of the unsupported arm falling back by its own weight during the anæsthetic and so stretching the circumflex (axillary) nerve. The arm can be supported by a nurse sitting at the head of the table, but it is a tiring task and as the operation

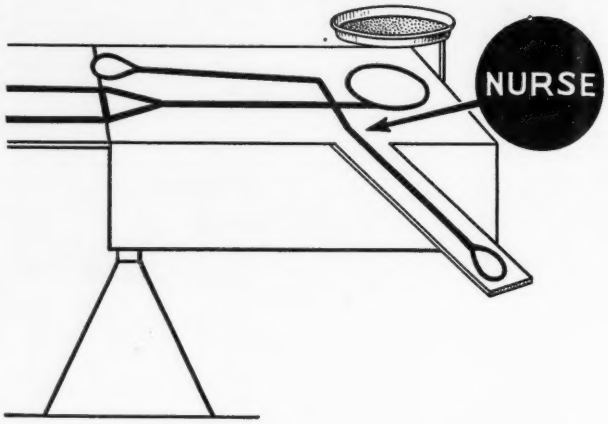


FIG. 164.—Drawing illustrating : (1) Sterilizable instrument tray which it is convenient to have placed in the position shown ; (2) Arm-board ; (3) Nurse's position at the head of the table which affords her a direct view (arrow) of the operation field.

proceeds she may from fatigue allow the arm to fall back. She is also very much in the way at the head of the table. A simple and satisfactory arrangement is to place the outstretched arm on an arm-board of the type employed by anæsthetists (*Fig.* 164).

Towelling.—The flank on the affected side is vulnerable from the point of view of towelling unless precautions are taken. The usual method is to roll the patient towards the unaffected side while the flank is painted with antiseptic and then to push a sterile towel into the space between the operating table and the patient's back. This towel, depending as it does on the patient's weight to keep it in position, is rather too easily displaced and if this should happen a gap is left posteriorly which is no longer sterile.

An alternative method is to sit the patient up—anæsthesia being maintained—while a mackintosh covered with a towel is introduced from the end of the table behind the whole of the patient's back down *as far as the buttocks* (*Fig.* 165). The back and flank of the patient on the affected side are then painted (*Fig.* 166) and when this has been completed the patient is gently lowered again into the horizontal position. While this is a little more trouble, it is a surer and more thorough procedure. Iodine should not be used as it is said to reduce the tolerance of the skin to X-ray therapy.

The Skin Incisions.—The skin incisions are planned in the usual way so that the growth is the centre of an elliptical island of skin which also always includes the nipple. In the average case the area

of skin removed allows closure of the wound with approximation of the skin edges without tension. Each case, however, will be assessed individually, and such factors, for example, as infiltration of, or adherence to, the skin will influence the operator towards a wider excision than in cases in which there is no attachment.

If we could be sure that X rays could ' sterilize ' the skin, then we might consider a more restricted removal of the skin. This assurance, however,

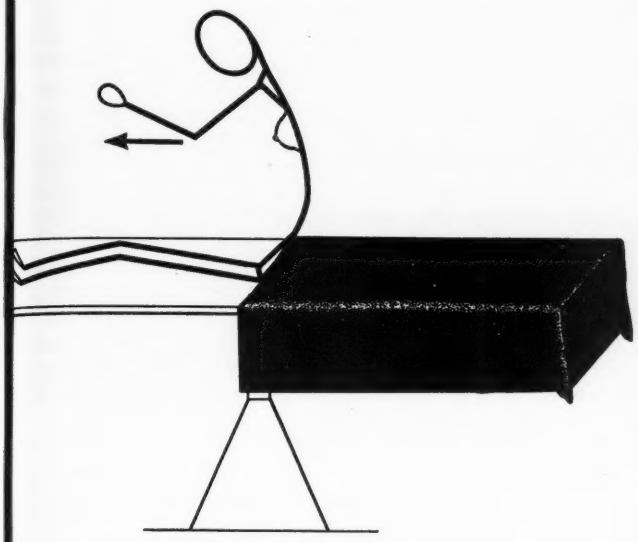


FIG. 165.—Towelling of the patient.

cannot be given yet, and in these circumstances an excision of skin clear of the tumour area must still be regarded as essential.

If the breast is very large and heavy, the usual elliptical incision is not satisfactory because it is inclined to leave folds of redundant skin in the flaps when these are brought together again. In my experience of these cases it is better to make the incision as if for a simple mastectomy (elliptical *oblique*) with a relieving incision towards the clavicle (*Fig.* 167).

The *lateral incision* is made first, its medial counterpart being outlined only at this stage by superficial marks upon the skin (*Fig.* 168). The lateral incision is made throughout its length from xiphisternum to the clavicle, but in the first instance should not cut deeper than the full thickness of the skin. Large veins course across the mamma immediately beneath the skin—that is to say, exactly ' subcutaneous ', and if they are divided, as they will be, by too bold a stroke of the knife, much unnecessary bleeding will be caused. A little experience is required to make this initial incision to the right depth and no farther. With practice the skin can be felt to split under the knife ; the precise amount of pressure exerted upon it being decided rather by the sense of touch than visually. Particular care is taken to place the clavicular end of the incision well to the inner side of the free margin of the anterior axillary fold (*Fig.* 168), or this part of the incision, when the greater pectoral has been removed, will result in the scar crossing the armpit, which later may seriously hamper the movement of abduction.

The *axillary* end of the flap is raised first. The incision is then deepened in this area until the muscular fibres of pectoralis major come into view. The elevation of the lateral flap is then gradually carried towards its abdominal end. When the whole of the edge of the flap has been raised, the dissection is continued on the flank in a backward direction until the red fibres of the anterior border of latissimus

FIG. 166.—The back and flank on the affected side are painted.

dorsi are exposed, and there it is stopped. Bleeding points, which are usually few in number, are tied by ligature. A warm *dry* pack is placed in the depth of the wound and the operator turns his attention to the clavicular end of the medial flap.

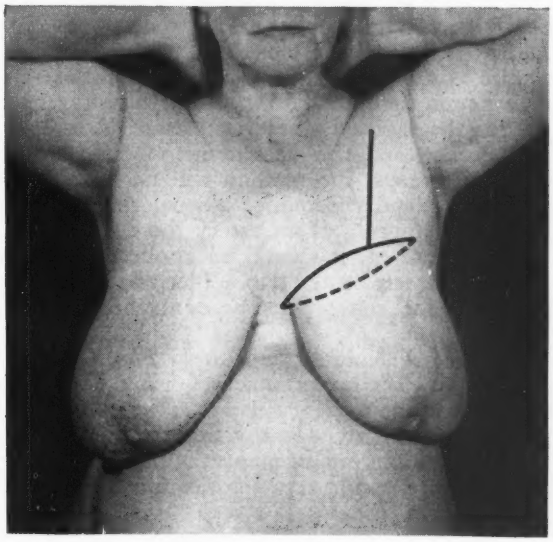


FIG. 167.—The incision used when the breast is heavy.

The Elevation of the Lateral Flap.—Before continuing with the general description of the operation, it may be useful at this stage to consider two matters which concern the lateral flap. These are: (1) the control of bleeding during its elevation; (2) certain errors associated with the elevation.

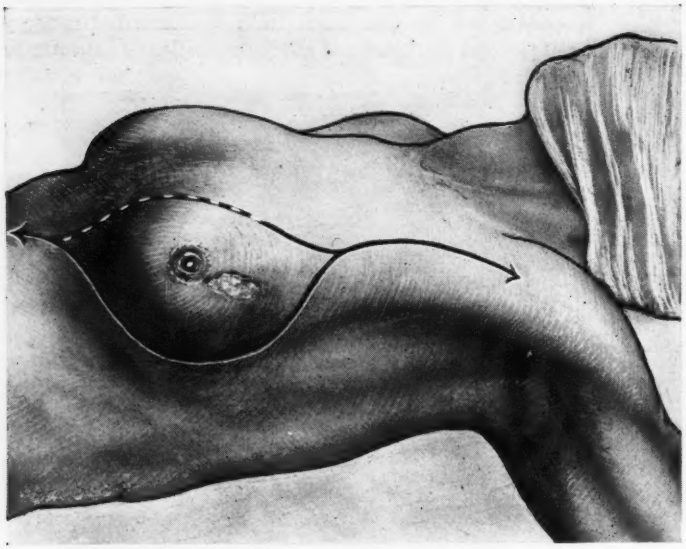


FIG. 168.—*The incision.* The axillary end of the incision is kept well to the medial side of the anterior axillary fold.

1. *The Control of Bleeding—'the Two-swab' Technique.*—Control of bleeding during elevation of the lateral flap is entirely by *swab pressure.* Artery forceps applied at this stage hold up the operation, get in the way of the dissection, and interrupt the even flow and rhythm of the whole procedure. The object is to suppress bleeding the instant it occurs, almost indeed before it occurs. The role of the chief assistant is as follows. Standing opposite to the surgeon the assistant works with a swab *in each hand.* As the elevation of the flap proceeds, so the assistant advances with his swabs keeping close to the point of the knife as it works its way posteriorly in the subcutaneous tissues. He stifles all bleeding from the breast and chest wall as it arises. With practice and the determination not to use artery forceps, the method will be found easy to apply, and will reduce blood-loss to an absolute minimum. If an artery forceps has to be made use of, it should be applied only to bleeding vessels on the flap. Bleeding from the breast side can always be controlled by pressure.

2. *Errors in Raising the Lateral Flap.*—It is a matter of great importance, requiring some skill and patience, to raise a flap of *even thinness* throughout its extent. In my experience this is the most difficult technical part of the operation and the following errors are common :—

Flap Too Thick :

a. Mammary remnants (*Fig.* 169): The line of cleavage between the breast tissue of the mamma proper and the subcutaneous fat is very definite, but it is not so obvious that pieces of breast tissue cannot be left attached to the skin-flap if this is cut too thick. This is particularly prone to happen if the dissection is carried out at a great pace and with little regard for bleeding. In these circumstances, the white lobules of the breast obscured by blood may be mistaken for fat and so cut through and left adherent to the deep surface of the flap, together presumably with any infected lymphatics which they may contain.

b. Dissection in the wrong plane (*Fig.* 170): The raising of mammary tissue with the flap is not only undesirable because it leaves the inner surface of the flap choppy and uneven, but because by cutting too thick a flap, the operator may be led into continuing the later part of the dissection on the *deep* face of the latissimus dorsi instead of superficial to it. In these circumstances, as well as failing to find the anterior edge of the latissimus dorsi for which he is searching, he is likely to be embarrassed by the plexus of large veins which occupy the space between the opposing surfaces of the serratus anterior and latissimus dorsi.

These veins, which are constantly present, have their origin on the chest wall and drain back to join the main subscapular vein. Injury to them sets up bleeding in the most inaccessible part of the wound.

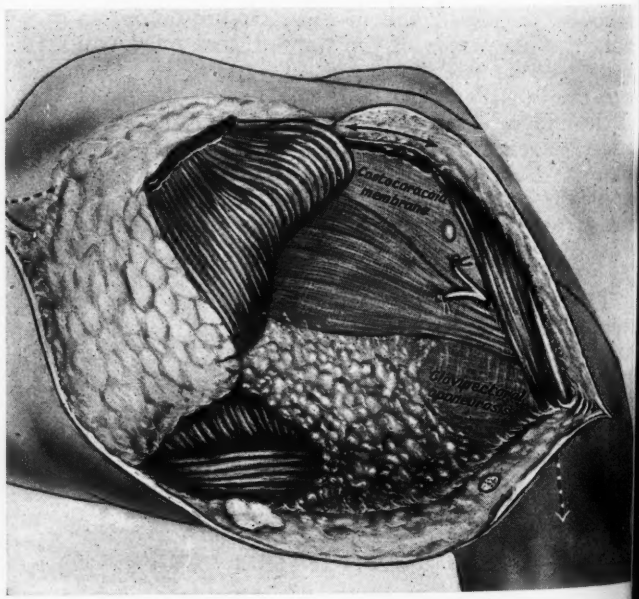


FIG. 169.—Drawing showing: 1, *The lateral flap.* If the flap is cut too thick, pieces of the breast may be left adherent to it; at the axillary end a whole lymph-node may be left embedded in the fat on the under surface of the flap. 2, The cut edge of the sternal portion of pectoralis major opposite the first two interspaces. 3. The two 'trunks' of the pectoral (thoracic) branches of the acromiothoracic axis. 4, The division of the bilaminar tendon of pectoralis major flush with the bone (arrow).

c. Lymphatic glands (*Fig.* 169): It is a simpler matter than one might suppose to leave infiltrated

lymphatic glands adherent to the inner surface of the lateral skin-flap at its axillary end. It is particularly liable to happen in fat subjects. Many so-called axillary '*recurrences*' can be attributed to the

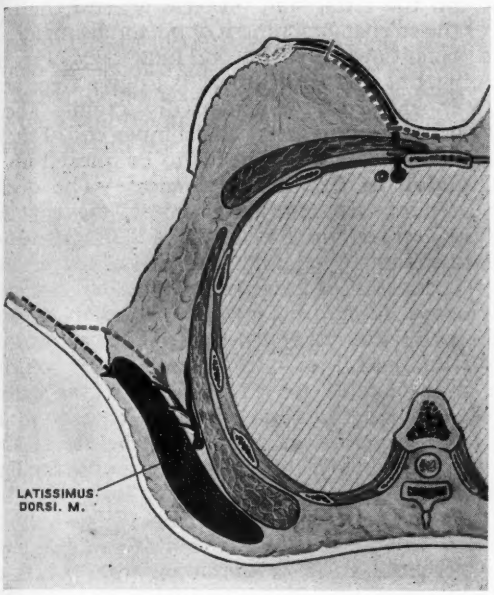


FIG. 170.—The diagram illustrates: 1, The elevation of the *lateral* flap in the correct plane (black arrow) and on too deep a plane (red arrow). 2, The elevation of the *medial* flap with the sites of division of the internal mammary vessels.

continued growth of glands left behind in this way. Like the mistakes referred to in the paragraphs above, this occurs most readily if the dissection is carried out on too deep a plane and if the visibility in the operative field is obscured by bleeding.

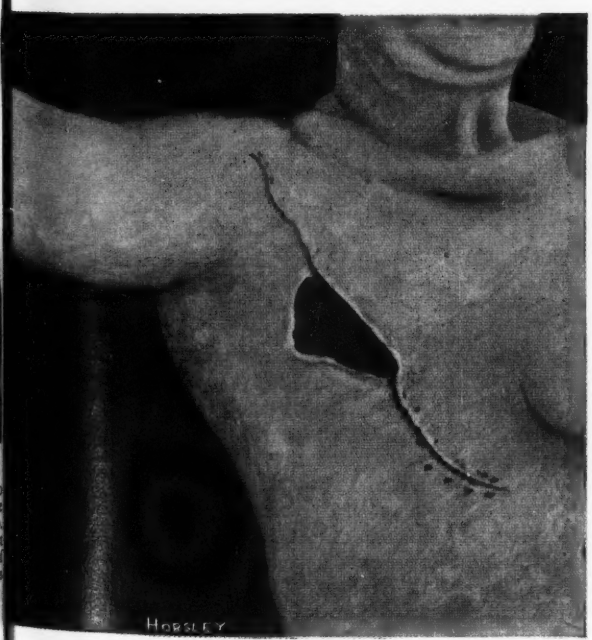


FIG. 171.—Marginal necrosis of the lateral flap, which delayed the start of post-operative X-ray treatment for 11 weeks.

Flap Too Thin:

Flap necrosis: The danger of flap necrosis is greatest in the *thin subject* and in the depth (i.e., the most posterior part) of the dissection. As the elevation approaches the region of the anterior border of the latissimus dorsi—it can happen, if the exposure is indifferent and the operator is in a hurry, that the dissection proceeds on too superficial a plane and the white (deep) under surface of the skin is laid bare or even buttonholed. If this should happen over too wide a stretch—particularly at the base of the flap—the blood-supply is cut off and gangrene of the flap will result. This may be widespread or only confined

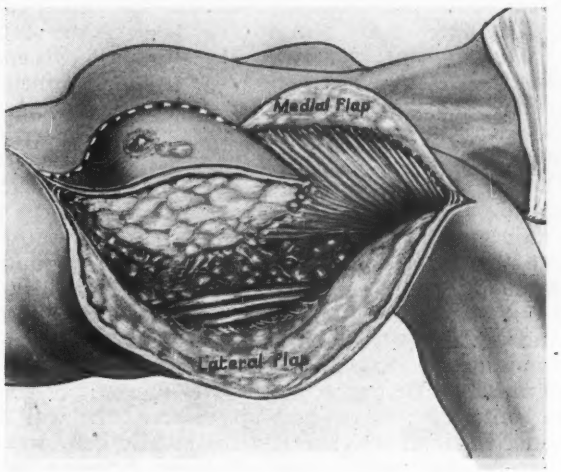


FIG. 172.—The flaps: The *lateral* flap has been raised to a point well beyond the anterior border of the latissimus dorsi; the *medial* flap has been raised at its axillary end only.

to the outer edge of the wound, but either way it is an unnecessary disaster, prolonging as it does the patient's stay in hospital and in many cases the commencement of post-operative radiotherapy.

All these points may seem trivial, yet if the blood-supply of a part of the flap is cut off by a careless stroke of the knife or over-heating with the diathermy, healing by first intention will not happen, convalescence will be prolonged by many weeks, the start of radiotherapy will be delayed, the final results so far as elasticity of the skin and range of movement of the arm are concerned will be impaired, and the patient herself will be worn out and depressed by an illness which she need never have experienced (*Fig.* 171).

The Medial Flap (Clavicular End) (*Fig.* 172).—The medial flap is raised at its *clavicular* end only. The skin over the greater part of the inner side of the breast is left undisturbed until the very end of the operation. (This method can be practised satisfactorily if the patient is thin and the breast of average dimensions, but in the stout patient, or if the breast is large, a better exposure will be obtained by raising the medial flap throughout its extent as in the classical descriptions of the operation.)

There are certain advantages in delaying the elevation of the medial flap as opposed to carrying this out as an initial step in the operation. Elevation of the medial flap involves division of branches and tributaries of the internal mammary vessels in

three separate situations: first, when the initial incision is made into the subcutaneous tissues, again as the base of the flap is approached, and again when the pectoral muscles are removed (*Fig.* 170).

By delaying the elevation of the medial flap until the perforating branches of the internal mammary have been secured: (1) *bleeding* is reduced and the flap can be raised avascularly; (2) *time* is saved because no vessels in the breast have to be ligatured or coagulated and few if any on the flap; (3) the *vitality* and warmth of the flap is preserved until the end of the operation.

The object in raising the clavicular end of the flap is to facilitate the detachment of the sternal fibres of the pectoralis opposite the first two spaces, and so improve the access to the apex of the axilla.

Division of the Pectoralis Major (Sternal Head).—Having raised the outer flap and sufficient of the medial flap to display the greater pectoral, the next step is to divide the main tendon of insertion of that muscle. A line of separation between the fibres of the clavicular and pectoral heads is determined, a finger inserted and the artificial gap so produced split down to the insertion of the muscle into the humerus. A finger is then passed under the main mass of fibres formed by the sternal head till it emerges on the far side. The bilaminar tendon of insertion is then divided. The division should take place close to the bone so that no stump of muscle is left attached to the humerus to form an unsightly bulge post-operatively. It will be noticed that the thickness of the muscle belly where it is divided varies considerably in different individuals, its bulk being related to occupation and right- or left-handedness.

Identification of the Acromiothoracic Vessels.—We are next concerned to find the pectoral (thoracic) branches of the acromiothoracic axis. These should first be sought where they lie upon the superficial surface of the pectoralis minor, somewhat hidden in a mass of loose white fascia. These vessels are then traced back to the acromiothoracic axis from which they arise, and which emerges over the upper border of the pectoralis minor, close to its coracoid attachment, where they are secured. They should be ligatured and not coagulated with the diathermy. It will be found that these branches give off a small leash close to their origin from the main stem vessel, which turns forwards into the substance of the pectoralis major and may easily be cut across unless it is sought out first and separately identified. The other three branches of the axis—acromial, clavicular, and humeral—all run in varying degrees in a direction away from the wound. They pass under cover of the clavicular portion of pectoralis major, and should as a general rule be left alone. Infected tissues rarely lie to the outer side of the axillary artery, so that dissection here in most cases is unnecessary. If it is undertaken by mistake, as for example when the line of separation between the sternal and clavicular portions of the muscle has been made too near the deltoid-pectoral interval, the difficulties of the dissection are considerably increased.

The Separation of the Sternal and Clavicular Portions of Pectoralis Major.—The separation of the sternal and clavicular portions of the pectoralis major, which so far has only been effected at the outer humeral end of the muscle, is now carried in the opposite direction as far as the upper end of the sternum (hard by the inner end of the clavicle). A finger is then passed beneath the muscle-fibres forming the highest part of the *sternal* attachment in front and the chest wall posteriorly, and the upper part of the sternal attachment of pectoralis major is divided, as far down as the second interspace (*Fig.* 169). The object of this detachment is to give freer access to the upper part of the axillary dissection. There is usually a perforating artery at the inner end of the first and second spaces which needs to be picked up.

Division of Pectoralis Minor.—The division of the tendon of pectoralis minor is the next step.

The Clavipectoral Fascia ABOVE *Pectoralis Minor.*—It will be remembered that the belly of the pectoralis minor muscle is enclosed in a sheet of fascia, the clavipectoral fascia, the upward extension of which is thin, like tissue paper, and called the costocoracoid membrane, and the lower extension of which is thick and aponeurotic, very much like the external oblique in the inguinal region. Owing to this difference in consistence between the fascia above and the fascia below the pectoralis minor, the finger is most readily introduced beneath its tendon from *above*, and this should now be done. To allow this finger to emerge on the lower side, an opening is made with the knife in the aponeurotic portion of the clavipectoral fascia, as illustrated (*Fig.* 173).

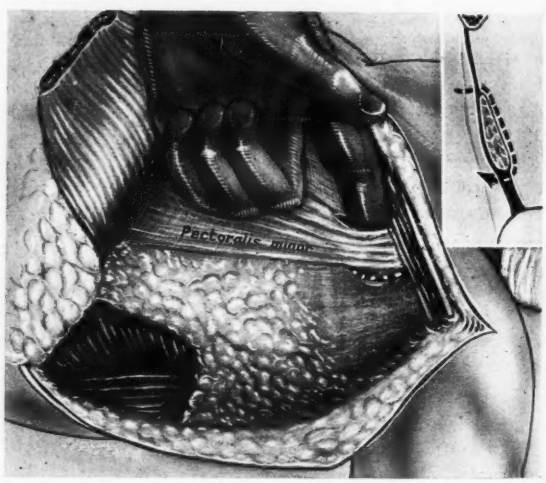


FIG. 173.—*The pectoralis minor.* The drawing and inset show the clavipectoral fascia, thin above the pectoralis minor (costocoracoid membrane) and aponeurotic below the muscle.

With the muscle stretched across the finger, it is most conveniently divided with curved scissors, close to the bone. There is frequently a small artery in the tendon of insertion.

The Clavipectoral Fascia BELOW *Pectoralis Minor* (*Fig.* 174).—The subsequent retraction of the pectoralis minor from the coracoid process artificially produces, on the distal side of the muscle, a fascial ridge which crosses the neurovascular bundle from side to side; this ridge, which is crescent-shaped, is continuous with the aponeurotic portion of the clavipectoral fascia, which in this situation is forming the downward expansion of the lower border of the muscle and is ensheathing the vessels.

To effect a clean dissection it is expedient to insert two fingers beneath the edge of this expansion and to separate it from the underlying neurovascular bundle. With the fingers in situ, and protecting the vessels, the outer edge of this fibrous sheet is divided from its attachment to the coracobrachialis on the outer side of the vessels, and then the line of separation curves inwards across the main vessels to finish on the medial side of the axillary vein immediately below the point of entry of the subscapular vein. With the completion of this step the axilla and its contents are laid open for the first time.

FIG. 174.—*The clavipectoral fascia.* The pectoralis minor has been divided; the retraction of the muscle produces a crescentic edge to the clavipectoral fascia as shown; the dotted line indicates the line of its detachment from the outer side of, and then across, the vessels.

The Axilla.—After division of the pectoralis minor it is a common practice to go straight to the apex of the axilla and begin the clearance of the subclavian vein. I prefer to precede this important part of the dissection by two preliminary steps.

The first is to define the tendon of latissimus dorsi right up to its insertion and so put an end to the dissection in the region of the axillary outlet at an early stage. The second is to free the axillary vein of its distal tributaries so as to facilitate the mobilization of its central end.

Definition of the Tendon of Insertion of Latissimus Dorsi (*Fig.* 175).—If attention is now directed to the region of the posterior boundary, it will be found that there is a *mass of tissue* (containing glands, fat, and vessels) *obscuring* the tendon of insertion of latissimus dorsi. This mass of tissue stretches from the lateral chest wall and lower border of the cut pectoralis major across the highest part of latissimus dorsi to the inner surface of the skin of the armpit and the adjacent segment of the axillary vessels.

It is convenient to divide this bridge of tissue forthwith. By so doing, the latissimus dorsi—muscle belly and tendon—will be displayed throughout its length. The definition of this muscle up to its tendon of insertion into the humerus marks the posterior limit of the reflection of deep fascia, and completes at an early stage of the operation the dissection in this area and adjacent axillary outlet.

The procedure is as follows:—

1. Display by dissection the main trunk of the subscapular vein for the last inch of its course into the axillary vein.

2. Pass a finger into the space produced by this dissection and direct it along the outer side of the subscapular vessels. In this situation the finger will be parallel with, and deep to, the anterior border of latissimus dorsi.

3. Divide the tissues now stretched across the finger. Some vessels running in the centre of the mass will need to be picked up between artery forceps.

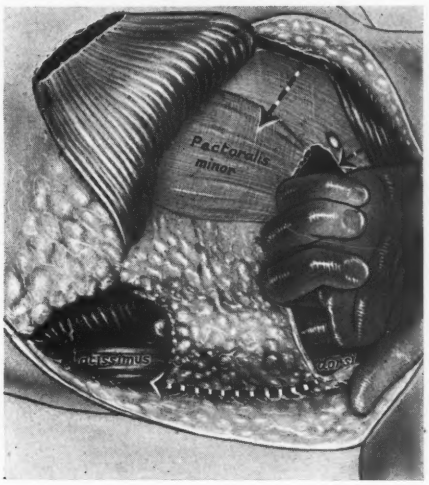


FIG. 175.—*The latissimus dorsi.* The reflection of the lateral flap exposes the anterior border of the latissimus dorsi in its lower two-thirds, but still leaves it covered in its upper third by a bridge of tissue which stretches from the chest wall across its tendon of insertion. The drawing shows a finger lying in contact with, but to the outer side of, the subscapular vessels. The bridge of tissue crossing the finger, i.e., all tissues to the outer side of the subscapular vessels, is divided. This leaves the anterior border of latissimus dorsi clear right up to its insertion into the humerus.

Care is taken to see that all glands and potentially infected tissues are displaced medially.

When this division has been completed, the anterior border of latissimus dorsi will be found to be clearly defined right up to its insertion.

Mobilization of the Axillary Vessels (*Fig.* 176).—The mobilization of the axillary vessels by division of their branches and tributaries is the next step. It is particularly important that the central end of the axillary vein should be freed from all tethering veins and nerves if a thorough dissection of the apex of the axilla is to be carried out. In my experience this high mobilization is most readily achieved by working in a *distal to central* direction (rather than the other way round), because the tributaries of the vein are long near the axillary outlet and therefore easily identified, whereas they become progressively shorter and more fragile as one approaches the central end of the vein.

Synchronously with the act of mobilization, the main vein should be cleared of all nodes and tissues which might be infected. Working with a dissector in one hand and gauze in the other, the tissues with any entangled glands are swept downwards *en bloc*. In stripping the tissues from the vein, the operator should push the *vein away* from himself with the

hand holding the dissector and draw the *tissues towards* himself with the hand holding the gauze. It is not so satisfactory to pull both the vein and the tissues in the same direction.

The Apex of the Axilla (*Fig.* 176).—Once the main axillary vein has been mobilized it should be rotated and retracted outwards so as to expose the tissues surrounding its central or subclavian end. This is the very summit of the axilla. It is the region of the *first rib*, the *first intercostal space* and the *first digitation of the serratus anterior*.

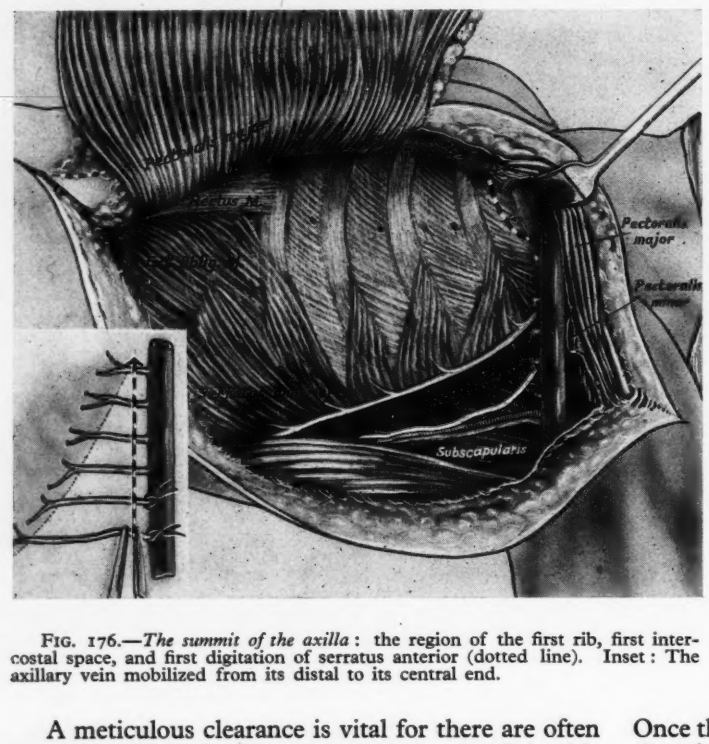


FIG. 176.—*The summit of the axilla*: the region of the first rib, first intercostal space, and first digitation of serratus anterior (dotted line). Inset: The axillary vein mobilized from its distal to its central end.

A meticulous clearance is vital for there are often minute *seedling glands* no larger than a lentil in this area. Only too frequently also there is a thickened infiltrated *lymphatic cord* lying to the inner side of the main vein, which needs to be broken off as high as possible. The importance of the dissection in this small triangle at the apex of the axilla cannot be exaggerated, and because the tissues are friable and the structures, particularly the subclavian vein (as it is here), vulnerable, the dissection cannot be hurried.

The Inner Wall.—The clearance of the medial boundary follows close upon the clearance of the apex of the axilla and is begun by detaching the uppermost fibres of the pectoralis minor from the chest wall. A finger or closed pair of dissecting forceps is next passed into the space thus produced, and, keeping in direct contact with the chest wall, it is directed posteriorly. The finger is worked up and down in this space when it will be found that a thick sheet of fascia is separated from the chest wall. Contained in this sheet of fascia will be seen the long thoracic nerve of Bell. It is a simple matter to dissect out and release the nerve—leaving the sheet itself in situ—until the fascia covering subscapularis, with which it is continuous, has also been separated.

The Posterior Wall.—Removal of the fascia covering the subscapularis is all that now remains to complete the dissection of the actual axilla. To effect this a pair of dissecting forceps is thrust, gently, through this covering layer, and into the small opening thus produced a finger is introduced. Particular care must be taken to pierce the fascia only. There are a number of small veins on the anterior surface of the subscapularis beneath the fascia, and if these are damaged the separation will be accompanied by free bleeding. The fascial sheet covering the muscle is now separated from the underlying subscapularis by a sweeping movement of the finger. It will be found that the fascial sheet from the chest wall and that from the subscapularis have a common line of attachment posteriorly in the groove between the chest wall and the inner or vertebral margin of the subscapularis. This double-layered sheet is now cut away from above downwards with a pair of scissors.

Lateral Chest Wall.—The axilla itself having been cleared, there remains only to separate a wide *ring of deep fascia* together with the underlying pectoral muscles from the chest wall. The general intention is to raise this mass towards the midline. The separation of the perimeter of this circle of deep fascia begins posteriorly by defining clearly, and throughout its length, the anterior border of the latissimus dorsi. The dissection then works forwards and inwards, separating, as it advances, the deep fascia from the anterior surfaces of the digitations of the serratus anterior, whose finger-like processes run forwards in line with the ribs. A sharp knife is needed, and care has to be taken not to carve pieces of the underlying muscle away. Once the dissection has reached the anterior extremities of the digitations of the serratus, the separation from the chest wall proceeds more easily.

As this mass is raised and rotated inwards, two sets of vessels emerging from the chest wall are encountered. The *first set* met with are placed well out on the lateral aspect of the chest wall (lateral perforating branches of the intercostal arteries). They are small but definitely recognizable, and emerge from each intercostal space approximately 5 in. from the lateral sternal margin. They can be and should be recognized and picked up. The *second set* of vessels are found—much more medially—one set to each intercostal space, and they emerge through the medial ends of the intercostal spaces at the lateral margin of the sternum. These are the well-known perforating branches of the internal mammary. They are large—particularly in the second and third spaces—and must be secured one by one. Identification of the 'perforators'—one set to each space—is made easier if the assistant opposite the surgeon raises the musculo-mammary mass vertically upwards from the sternum. Each set of vessels is separately identified by dividing the innermost fibres of the pectoralis major which overlie them, and they are secured and then divided between artery forceps. When this has been completed

the whole mass in the assistant's hand is returned across the chest. We are now ready to carry out the final act of the operation—the elevation of the medial skin-flap.

The Medial Flap (*Fig.* 177).—The skin incision over the inner part of the breast—where it had not previously been deepened—now has this carried out in the ordinary way. A thin flap of skin and subcutaneous tissue is elevated as far as the *midline* of the sternum. This manœuvre is almost avascular and rapidly effected, since the perforating vessels which carry the blood-supply to the flap have been secured at the previous stage. The mass is now only left attached to the thorax by a thin linear strip of muscle and fascia up and down the sternum. With a few touches of the knife these remaining fibres of the pectoralis major are divided together with the upper part of the anterior rectus sheath in lower hemisphere growths. The separated mass, which consists of the mamma, the underlying pectoral muscles, and all glands and infected and potentially infected tissues *en masse* can now be discarded.

Hæmostasis.—There only remains to ensure absolute hæmostasis over the whole operation area. A number of bleeding points will require attention. For the first time in the operation a hot pack should be applied—for hæmostatic purposes. All the larger vessels should be tied, although many of the smaller ones can safely be sealed with diathermy. Our usual procedure is: (1) To ligature the perforating branches of the internal mammary at the outer border of the sternum. These vessels are too large to be coagulated by diathermy with safety. (2) To ligature the venous tributaries of the subscapular vein as they run perpendicularly backwards far out on the flank. (3) To coagulate with diathermy the small vessels lying intermediate between the other two groups.

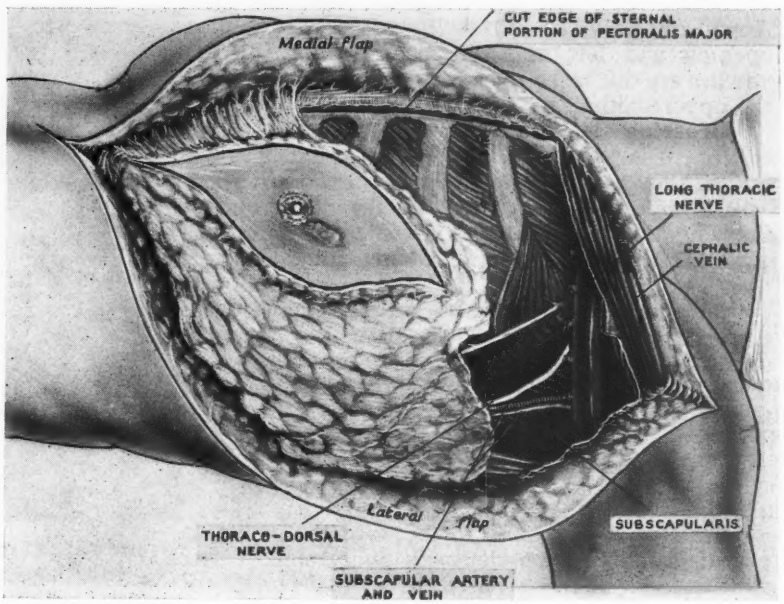


FIG. 177.—*The last stage.* The drawing shows the breast about to be separated finally from the chest wall by the division of the few remaining fibres attaching it to the sternum. The cut edge of the pectoralis major (sternal attachment) is shown, but in practice the separation, which extends to the midline of the sternum, actually removes these fibres also. In the drawing can also be seen upon the chest wall the perforating branches of the internal mammary artery (ligated) and the lateral branches of the posterior (aortic) intercostals (coagulated) and the long thoracic nerve of Bell. Upon the subscapularis the nerve to latissimus dorsi is seen conspicuously separated from the subscapular vessels, which run together. The cephalic vein and stump of the pectoralis minor can also be seen.

Closure.—The two flaps are approximated in the first instance by a series of stay sutures placed close to the skin edges. None of these stitches is inserted more than half an inch from the skin edge. Tension sutures as such are never used. It does not appear to matter greatly how the final edge-to-edge closure is made—whether it be with metal clips or a continuous type of blanket stitch reinforced with interrupted sutures; we have not found that the continuous suture has any special advantage and it is somewhat tedious to remove.

Failure to close: Inability to get the skin edges together is not a serious matter. It is wiser to leave a gap—usually in the centre of the wound—than to try and approximate the edges by force. *Tension sutures should never be used.* Flaps forcibly pulled together are an immediate cause of shock. Later when the tension sutures are removed the skin edges will separate, sometimes widely, so that healing by granulation is as inevitable as in those patients in whom no attempt has been made to draw the edges together, but with the difference that the separation is more widespread. If a gap is left deliberately the skin edges should be sewn down with a few catgut sutures to the underlying intercostal muscles; this prevents retraction. The raw area can be immediately grafted or it can be left to granulate. There is not a great deal to choose between the two alternatives. The decision will depend upon the patient's condition at the end of the operation, with a bias towards immediate grafting if this seems safe.

Drainage.—All wounds should be drained, however dry they may appear at the end of the operation. The closure of wounds without drainage results in a collection of fluid beneath the flaps in a high proportion of cases. These collections, it is true, can be aspirated, but if overlooked they may become tense and threaten the blood-supply of the flaps. Repeated aspirations offer an opportunity sooner or later for the introduction of infection, which quickly becomes widespread and may cause severe constitutional illness.

Site of Drainage.—The three commonest sites for drainage are: (1) from the *epigastric* end of the incision; (2) from the *axilla* by stab incision; and (3) from the *flank* by stab incision.

Most surgeons employ only one of these drainage openings, others employ a combination of them. All are equally effective, but one—the axillary—is more uncomfortable than the others. In this series closed axillary drainage, by a stout tube and a long

length of Paul's tubing reaching to a bottle on the floor, was used in the first 63 operations, but after this it was given up, not because it was ineffective, but because the patients complained of discomfort from the thick-walled tube. It is a very clean method but in my experience the sinus left after its removal is often painful and slow to heal. A more potent argument against axillary drainage is the quality of the skin in this area, which is *hair bearing* and freely endowed with *sweat glands*—glands which from experience we know do not contribute to healing by first intention.

Epigastric or flank drainage was used in the remainder with corrugated rubber dam, three corrugations wide. *Flank* drainage—that is to say drainage by a stab incision through the centre of the lateral flap—has the advantage that the main wound can be *sealed off completely* from the drainage opening and left undisturbed until the clips or stitches are due to be removed. This should reduce the opportunities for cross-infection.

Dressing in the Theatre.—*Mastisol* or its equivalent is painted on to the skin on either side—but not over—the whole length of the incision. Special care is taken to seal off the upper end of the wound in the *shoulder* region where the dressings are liable to slip and expose a bare area. Gauze is then applied to the wound in the usual way, followed by cotton-wool; a separate loose pad is placed in the axilla. The area round the drainage opening is similarly treated, omitting the mastisol. The whole is then enclosed in a many-tail bandage, not too tightly applied. The arm is placed in a sling.

POSTOPERATIVE MANAGEMENT

Nursing

Position.—On return to the ward the patient is placed flat until consciousness is regained, after which she should be raised to a semi-recumbent position, with the affected arm in a sling and supported on a pillow. Repacking of the drainage area with sterile wool during the first 12 hours after operation may be required on several occasions.

Care of the Wound.—

1st, 2nd, and 3rd days:—

Dressings: The first dressing is done on the morning following the operation. The early removal of *wet* dressings makes the patient more comfortable. The area of the drainage-tube *only* is exposed and the main mastectomy wound is left undisturbed.

Drainage rubber: the drainage rubber is shortened by 2 in. on each of the first two days and removed on the third day, unless there is still a free discharge. After the third day drainage is more of a liability than an advantage and should be dispensed with as soon as possible even at the risk of a pocket of serum collecting. It is a matter of experience that if drainage is too prolonged, the main wound is likely to become infected as a result of the frequent changes of dressing in this area.

5th day: the whole wound is exposed for the first time, and the *alternate* clips or stitches are removed.

7th day: the remaining clips and stitches are taken out and a dry dressing applied. As soon as the wound is firmly healed, vaseline is applied to the scar, and the dressings omitted except for a layer of wool which some patients like to keep for warmth and protection.

8th day: the patient is usually sufficiently recovered to get up in a chair.

10–14th day: During this period, that is, as soon as the wound is healed, the patient is ready for post-operative irradiation provided this can be carried out as an *in*-patient. She will not be strong enough to attend as an *out*-patient until three to four weeks after leaving hospital.

Care of the Arm.—For the first two nights following the operation the mastectomy patient sleeps more comfortably if the arm is allowed to remain by the side. On the *second* morning, however, the sling is removed, and the arm abducted

Fig. 178.—Care of the arm: the position from the second morning onwards.

and kept at rest on a pillow until the tenth day, when gentle massage and movements are commenced (*Fig.* 178). Active movements before this time are to be *discouraged*. In my experience too early movement has caused separation of the skin edges of the flaps and increased the liability to serum collection, without any compensating advantage in the ultimate range of movement at the shoulder-joint, which in the majority of cases, unless there has been skin loss, is extremely satisfactory.

OPERATIVE MORBIDITY

Recovery from the operation with healing of the wound by first intention is the desired objective. This should be complete by the tenth post-operative day. Prolongation of the recovery phase beyond the tenth post-operative day is most commonly attributable—apart from respiratory complications—to one of three local setbacks: flap necrosis, wound sepsis, or a collection of serum.

Flap Necrosis (*Fig.* 179).—Flap necrosis, and its more limited form, *marginal necrosis* or sloughing of the skin edges, is the most serious of the commoner complications of radical mastectomy. It is very common. It delays healing and by so doing on the one hand greatly prolongs the patient's stay in hospital and on the other postpones the commencement of post-operative X-ray therapy should this be intended. It is a complication which is largely preventable. It is due to interference with the

blood-supply—usually of the lateral flap—and this can be brought about in several ways.

1. *Flap too thin*: The flap may be devitalized by raising too thin a flap. As has been pointed out, thinning of the flap beyond the safety margin is particularly liable to happen in the region of its base, which being in the depth of the wound is the most difficult part of the dissection to control accurately.

2. *Flap too hot*: The flap may be devitalized by overheating. If diathermy is used to raise the flap

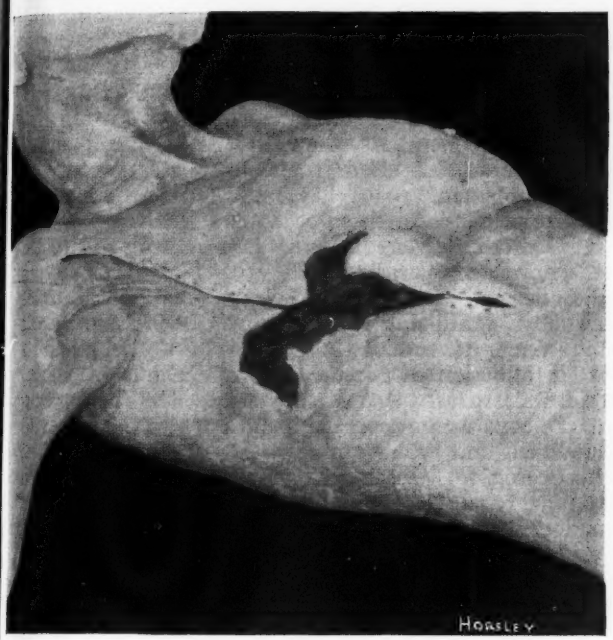


FIG. 179.—Flap necrosis (both flaps), resulting from the use of tension sutures.

very considerable heat is generated and the concentration of heat is maximum along the line of attachment of the base of the flap.

3. *Flap too cold*: If one or both flaps are fully reflected at the beginning of the operation, their vitality may be endangered unless precautions are taken to avoid undue exposure to cold. A common practice is to apply heat through the medium of hot wet packs constantly changed. This is not only time-consuming and interrupting, but liable to be inefficient, because the hot pack, if forgotten, quickly becomes a cold one. It is more satisfactory as previously suggested to use a thick pad of dry gamgee or an abdominal type of dry pack recently sterilized in the autoclave, so that it is still hot from the baking.

4. *Too much tension*: If the skin edges are forcibly drawn together with tension sutures a part of the blood-supply of the flaps is liable to be cut off.

5. *Too tight bandaging*: If a many-tail or other bandage is applied too tightly at the end of the operation not only will the breathing be seriously embarrassed but the actual blood-supply of the flaps may be endangered.

6. *Too soon after irradiation*: If an incision is made into irradiated tissues before they have had time to recover their natural healing qualities the suture line may break down.

Wound Sepsis.—*See* Measures against Infection.

Collection of Serum.—A mixed collection of serum and lymph varying in extent is not uncommonly seen. It is often incorrectly referred to as a hæmatoma, which in my experience is very rare. The common causes of such a collection are incomplete hæmostasis, overuse of the diathermy, omission of drainage, and too early post-operative movements. This collection of serum may occupy the whole flank, or it may be, and commonly is, confined to the region of the *axillary end* of the incision. It disappears with aspiration. It should not be left to absorb on its own, as if tense it may interfere with the blood-supply of the flaps.

SUMMARY

The operative mortality of radical mastectomy in the best hands is low, but there is still a higher operative morbidity than one would expect to find after half a century's experience of an operation.

Improvements in the general management of radical mastectomy, perhaps because of its relative antiquity and the tendency to familiarity that goes with this, have not kept pace with the advances that characterize the more confined specialties. For this reason it is inclined at the present time to compare unfavourably with procedures of equal magnitude performed in the special departments. It is important for the reputation of general surgery that it should not be possible to level such a criticism. Measures aimed at raising the general standard and conduct of the operation of radical mastectomy are described.

I am very much indebted to the chief assistants, house surgeons, and theatre staff who helped with these operations; to Dr. Ernest Landau, who gave the majority of the anæsthetics; and to Mrs. Blackburn for her careful drawings made during the operations.

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ARGENTAFFINOMA OF THE GASTRO-INTESTINAL TRACT

By DOUGLAS R. K. REID

ARGENTAFFINOMA, or carcinoid tumour, is best known as a solitary and invariably benign growth of the vermiform appendix. In other sites the tumour is uncommon, but of considerable interest in view of the fact that it is not infrequently associated with severe symptoms, and is found to be malignant in a high proportion of instances.

The object of this paper is to present 25 cases of argentaffinoma, including 8 which were extra-appendicular in site. Of these, 6 have caused major abdominal symptoms and 2 have resulted in death.

HISTORICAL

Lubaarsch (1888) is credited with the first description of argentaffinoma. He recorded two cases involving the ileum, and regarded them as atypical carcinoma, of which the distinguishing features were multiplicity, lack of glandular structure, and absence of metastases. In 1890, however, Ransom reported a similar case with metastasis to the liver, and Nothaft in 1895 described secondaries in lymph-nodes.

Argentaffinoma was first clearly separated from adenocarcinoma by Oberndorfer (1907), who employed the term ' carcinoid '—i.e., little carcinoma—although he regarded the growth as essentially benign.

Interest was at first centred mainly in the histological features of the tumour, and many theories were advanced regarding its histogenesis. Glazebrook (1895) and Kelly (1908) described it as an endothelioma. Trappe (1907) postulated an origin from pancreatic rests. Following this lead, Toniessen (1909) suggested that these hypothetical rests comprised cells analogous with those of the islets of Langerhans, while Saltykow (1912) maintained that they were identical with islet tissue. Bunting (1904), Burckhardt (1909), and Krompecher (1919) regarded argentaffinoma as a basal-cell carcinoma of mucous membrane analogous with that of the skin. Ehrlich (1928) believed it to be derived from Auerbach's plexus, and referred to it as " immature sympathetic neurocytoma ", while Aschoff (1939) described it as a nævus of mucous membrane. Engel (1923) and Primrose (1925) returned to the concept of embryonal rests.

In 1910 Huebschmann suggested the possibility of an origin from the ' yellow cells ' described by Kulschitsky (1897) and Schmidt (1905). These are now generally known as the Kulschitsky cells of the crypts of Lieberkühn, and their identity with the cells of carcinoid tumours was finally demonstrated by Gosset and Masson (1914), largely on the basis of their affinity for silver stains.

THE ARGENTAFFIN CELL

Kulschitsky cells are found chiefly in the depths of the crypts of Lieberkühn. They are most numerous in the appendix, where they number from 5 to 10 per crypt. In the small intestine they are distributed most abundantly in the duodenum (Maximow and Bloom, 1944) and are less numerous in the jejunum and ileum. They are found in small numbers in the stomach and colon.

The cells are found singly, lying on the basement membrane, although some also establish contact with the lumen of the crypt. They tend to be pyramidal in shape, with the narrow end directed inward. The nucleus is large, round, or ovoid, and possesses a distinct nuclear membrane. The cytoplasm may contain lipid vacuoles or granules: the latter are distributed chiefly between the nucleus and the basement membrane, and have an affinity for various chemicals, including eosin, osmic acid, and silver. Not all of the cells possess granules, and Erspamen (1939) has indicated the existence and importance of these agranular forms.

Gosset and Masson (1914) believed the cell to be of endodermal origin, Kull (1925) regarded it as mesodermal, and Danisch (1924) as ectodermal. Equally conflicting views have been expressed regarding its function. Ciaccio (1905) suggested the production of adrenaline, while Danisch (1924) and Raiford (1933) described the cell as belonging to the chromaffin tissue, in common with the adrenal medulla and the sympathetic nervous system. According to Masson, the argentaffin cells form in the aggregate a gland, the function of which is endocrine and related to the submucous nerve plexus.

Popoff (1939) has advanced a new hypothesis in which the argentaffin cell represents a stage of rest or functional exhaustion in a cycle of changes through which the mucus-secreting intestinal cell is constantly passing. The possibility has also been suggested that the argentaffin cell may secrete the intrinsic factor of Castle (Eros and Kunos, 1933, Jacobsen, 1939).

INCIDENCE OF ARGENTAFFIN TUMOURS

Location.—According to Raiford (1933), 85 per cent of argentaffinomas occur in the ileocæcal region, and of these the great majority are found in the appendix. Porter and Whelan (1939) give the incidence of appendicular carcinoid as 0·5 per cent of all appendices examined pathologically. It is within the bounds of possibility that this predilection of carcinoids for the appendix is more apparent than real. The appendix is the most commonly excised abdominal organ, and its pathological aberrations can hardly escape notice, whereas similar conditions in other parts of the gastro-intestinal tract may well remain undiscovered throughout life.

Argentaffin tumours have been found with decreasing frequency in the ileum, jejunum, duodenum, colon, and stomach. Elsewhere they are extremely rare, although Willis (1940) has suggested the possibility of primary occurrence in the pancreas, while Joel (1929) and Porter and Whelan (1939) each report a case of argentaffinoma of the gall-bladder. An even more exotic site has been recorded by Stewart, Willis, and De Saram (1939), who described two cases of argentaffinoma originating in ovarian teratomas.

Considerable attention has been paid in the German and American literature to carcinoid tumours of the small intestine. Such cases have been collected from the literature by Cooke (1931), Humphreys (1934), Ariel (1939), and Dangremond (1942). The last author was able to gather a total of 283, including 2 personal cases. Since 1942 a further 33 examples have been added, apart from the 30 reported by Docherty, Ashburn, and Waugh (1943), as having been found in the Mayo Clinic in the period 1906–1943.

Thus over 300 carcinoids of the small bowel have been reported to date. Of these the great majority have been found in the ileum, and to a less extent in the jejunum. The duodenum is comparatively rarely involved, in contradistinction to the large numbers of argentaffin cells which are said to occur there. A Meckel's diverticulum has been the site of argentaffinoma in 5 instances recorded in the literature.

The tumour is uncommon in the large bowel or rectum. Searching the literature in 1942, Stout was able to find records of 4 carcinoids of the cæcum, 10 of the colon, and 6 of the rectum. To these he added 6 rectal cases. Potter and Docter (1944), Nelson (1939), and Snyder and Waugh (1941) each added a case of carcinoid of the cæcum, while Dukes (1945) refers to 9 such tumours situated at the recto-anal junction. Thus at the present time a total of 7 cæcal, 10 colic, and 21 rectal carcinoids have been recorded.

Only 6 carcinoids of the stomach can be found in the literature: these have been reported by Masson and Martin (1928), Feyrter (1934), Raiford (1933), Bailey (1934), and Porter and Whelan (1939). The stomach must therefore be regarded as one of the least common sites of argentaffinoma.

Sex.—Carcinoid of the appendix is more common in females than in males in a ratio of 3 to 1 (Moore, 1938). A similar distribution has not generally been noted for extra-appendicular growths, which have been reported in male and female equally. In the present series of 8 extra-appendicular cases, however, there are 6 females and 2 males.

Age.—The age distribution of patients with argentaffin tumours is of considerable interest in relation to their site. Most authors give the average age for appendicular cases as 25, and for the extra-appendicular growths as 57 years (Ariel, 1939). The inference may be that most of these tumours, irrespective of site, are present from a comparatively early age, and that the appendicular growths come to light in the first half of life by reason of the correspondingly high incidence of appendicitis in young people. This concept is consistent with the known fact that argentaffin tumours grow exceedingly slowly.

Race.—The tumours have been found in both white and negro subjects. There is no known predilection for any particular race or colour, although most cases have been reported from German or American sources.

PATHOLOGY

The pathological features of argentaffinoma are highly characteristic, and allow of little possibility of confusion with other conditions. It is the commonest tumour of the appendix, and according to Raiford (1933), accounts for 8·3 per cent of neoplasms of the small bowel.

The tumour is always primarily submucous, and is usually small, i.e., ½ to 2 cm. in diameter. The largest examples on record are those of Bretschger (1937) and Dangremond (1942), which were respectively the "size of a fœtal head" and 10·5 cm. in diameter.

Firm, hard, or rubbery in consistency, the tumour may be greyish-white in colour or it may possess the distinctive yellow colour which is due to the presence of large amounts of intracellular cholesterol. Necrosis and hæmorrhage into the tumour tissue are practically unknown.

Although multiplicity is regarded as being a typical feature of argentaffinoma, the appendicular growths are invariably single, while only one-third of the extra-appendicular cases are multiple. In these the usual number of nodules is from 2 to 10, although as many as 68 have been found in the small intestine. Of the few stomach and colon cases recorded, the majority have been single.

FIG. 180.—*Case* 19. Argentaffinoma of the ileum. Note the extreme degree of invagination of the muscle wall, which is well preserved. Tumour tissue extends from the submucous to the subserous layers. (× ⅔.)

Ulceration is said by most authors to be rare, but Docherty et al. (1943) state that it is quite common to find shallow, small, fibrotic ulcers, which represent merely a superficial loss of epithelium over the tumour.

There is no true capsule, although in many cases the growth is clearly circumscribed and limited to the submucosa. In others it is seen to invade the muscle coats, which are as a rule, however, well preserved, and it may extend into the mesentery.

In the appendix, the tumour takes the form of a bulbous nodule, or less frequently a submucous plaque; it may project toward the lumen, and assume a central position. Seventy per cent lie at the tip, and very rarely is the proximal half of the organ involved.

In the small bowel, a purely submucous tumour may be sessile or polypoid. When it invades the muscle coats, a highly characteristic feature consists of buckling of the bowel wall (*Fig.* 180), with resulting invagination of the tumour toward the lumen. This distortion is due to contraction of the fibrous and abundant elastic tissue of the stroma, especially in the subperitoneal coat, and it constitutes the mechanism whereby even very small carcinoids may produce intestinal obstruction. The growth may

assume an annular form, although it does not as a rule completely encircle the bowel wall; this type produces obstruction partly by stenosis and partly by buckling. Intussusception has resulted from the presence of a polypoid tumour, and in other cases obstruction has been the result of adhesion of coils of bowel in the vicinity of a carcinoid, particularly when this has invaded the mesentery.

Microscopic Features.—The cells are remarkable for their regularity of size and appearance. They are small, polygonal, or ovoid in shape, with large round central nuclei. The cell membrane is ill defined, and the cytoplasm moderate in amount. It may contain lipid vacuoles or argentaffin granules: non-granular forms occur, and carcinoid tumours composed entirely of non-granular cells have been described (Stout, 1942). The nuclear membrane is distinct, and the nucleus tends to be clear and vesicular, with numerous amitotic figures. Mitosis is extremely rare, even in growths which have metastasized. A variable number of cells may be pyknotic, but necrosis is never seen, except as the result of inflammation and suppuration.

The cells are compactly arranged in alveoli, strands, and islets. Palisading may be observed in the peripheral cells of the alveolus, which assume a cylindrical form. Small groups of racket-shaped cells may arrange themselves in rosette fashion around a drop of eosinophilic hyaline material, but there is no true acinus formation.

The stroma consists of trabeculæ of fibrous tissue separating the cellular alveoli. In appendicular carcinoids it is dense and abundant as a rule, but this is not always true of the extra-appendicular tumours, in which the trabeculæ may be very fine.

The presence of hypertrophied smooth muscle in the stroma has long been recognized as characteristic. Masson drew attention to the presence of neurinomatous elements, while Cooke (1931) has emphasized the abundance of elastic tissue. Cellular infiltration of the stroma is not marked, although eosinophils are found, especially in appendicular tumours.

Metastatic deposits closely resemble the primary growth, but the stroma tends to be scanty, and lacks muscle and nervous tissue.

Malignancy.—All argentaffinomas have the microscopical structure of carcinoma simplex, and in all, whether ' benign ' or ' malignant ', the cellular features are constant. It is, therefore, not possible to distinguish the benign and the malignant on histological grounds alone; indeed, it is probably sounder to regard all carcinoids as at least potentially malignant, and to classify them according to their behaviour, as localized, invasive, or metastasizing.

The smallest argentaffinomas are confined to the submucosa, but frequently otherwise benign growths infiltrate the muscle coats of intestine or appendix, and groups of tumour cells are commonly found in the subserosa. Malignancy in the form of local invasion of the mesentery or of actual metastasis is extremely rare in the appendicular carcinoid, only 17 such cases having been recorded (Docherty, Hopping, and Masson, 1942). Extra-appendicular tumours, on the other hand, occurring at a later period of life, are much more prone to display obvious malignancy. Thus out of 283 cases of argentaffinoma of the small bowel collected up to 1942, 81 (or 28·6 per cent) were malignant. Out of 38 growths of the colon and rectum, and 6 of the stomach, respectively 8 (21·1 per cent) and 2 (33 per cent) have metastasized. The highest rate of malignancy has been observed in the cæcum, with 5 malignant out of 7 recorded cases.

It has been stated that argentaffinomas metastasize only after attaining a relatively large size (Humphreys, 1934). This, however, is not always true: for example, of the 2 cases reported by Dangremond (1942), one was very large and had not metastasized, while the other was a small submucous nodule, and had spread to the regional lymph-glands.

Metastasis takes place chiefly to the lymph-nodes of the mesentery, of which one or many may be involved. Secondaries have been found in the liver, and in a few instances in the retroperitoneal tissues, lung, pleura, brain, spinal meninges, pancreas, and inguinal lymph-nodes. Pelvic masses and diffuse peritoneal carcinomatosis are not common.

Both primary and secondary tumours grow very slowly, and cases are on record in which secondary deposits have been observed over periods of years, without apparent progression.

It would appear that the high rate of malignancy in the extra-appendicular as opposed to the appendicular cases is related to the age incidence in the respective groups, and that this in turn merely reflects the length of time the tumour has been present. It has been stated by Gaspar (1930) that all carcinoids are potentially malignant, and that local invasion or remote metastasis is only a matter of time. In this connexion it is interesting that the only carcinoid of the appendix to reveal invasion of the mesoappendix, in the present series of cases, occurred in a patient aged 71.

CLINICAL FEATURES

The relationship between carcinoid tumour of the appendix and appendicitis is not clear. It is obvious that when a carcinoid is situated in the middle or rarely the proximal third of the appendix, it may and frequently does act as a mechanical obstruction, and lead to acute suppurative or gangrenous appendicitis. The association, however, of acute inflammation with a carcinoid in its common situation at the tip of the organ cannot be explained on such mechanical grounds, and in the present state of our knowledge is to be regarded as probably fortuitous.

When an appendix is removed on account of chronic pain of apparently appendicular origin, and a carcinoid is found, the relationship is obscure. Masson (1930) formulated the theory of " neurogenic appendicitis ", in which the essential and primary lesion was stated to be proliferation of nerve tissue in the form of small neuromas. Secondary infiltration of nerve-sheaths by argentaffin cells then led to the development of carcinoid. According to this view, the tumour is the result of chronic appendicitis, but not in itself the cause of appendicular symptoms.

Extra-appendicular argentaffinomas might be similarly explained as secondary to chronic inflammatory changes, but in them neural hyperplasia is much less consistently found, and they are better regarded as true tumours (Porter and Whelan, 1939).

They are in a large proportion of cases asymptomatic, and may remain so even up to the point of widespread metastasis. On the other hand, they may cause acute or chronic intestinal obstruction, and this tendency is greatly enhanced in the malignant growths. Humphreys (1934) found that 24·4 per cent of small-bowel carcinoids were associated with obstructive symptoms. Cooke (1931) showed that out of 83 cases of simple argentaffinoma of the small intestine 6 were found at operation for obstruction, while 10 out of 21 malignant growths had caused obstruction.

Recurrent attacks of colicky pain and vomiting, associated with a history of flatulent dyspepsia, constitute a common set of symptoms, and may suggest the erroneous diagnosis of cholelithiasis. Bleeding from the bowel is uncommon, owing to the infrequency of ulceration, but diarrhœa is a very constant and unexplained symptom. It may alternate with constipation, and may occur both during and between attacks of pain.

Debility with loss of weight only occurs with the onset of remote metastases, and even in their presence may be long delayed.

A palpable mass may be found, and may consist of the primary tumour, but more frequently it represents a mass of secondary growth, distended and hypertrophied bowel, or coils of bowel matted together by adhesions.

Radiological study of the small intestine after the administration of barium may reveal the characteristic appearance of acute angulation of a loop, with stenosis or a filling defect at the apex. (Miller and Herrmann, 1942.)

The duration of symptoms is of importance, ranging from six months to several years. No case has been diagnosed pre-operatively, and, indeed, the diagnosis at laparotomy may be difficult. It is suggested by the presence of single or multiple nodules of the small bowel, with or without secondary growth in the mesentery, lymph-glands, or liver, and accompanied by minimal symptoms of maximal duration.

MATERIAL

A search of the pathological material of the Dunedin and Auckland Hospitals has brought to light 25 cases of argentaffinoma (*Table I*). The site of the tumour is in 17 instances the appendix, in 6 the ileum, and in 1 each the stomach and colon. The 8 extra-appendicular tumours have all occurred within the last ten years (1937–47), and 3 of them have come within the personal observation of the author.

Among the 17 appendicular cases there were 11 females and 6 males. The youngest patient in this group was 9, and the oldest 71, the average being 30·1 years.

Table I.—Details of 25 Cases of Argentaffinoma

Case	Name	Age	Sex	Location of Tumour	Infiltration	Metastasis	Symptoms
1	M. T.	21	F.	Appendix	Muscle	—	Not known
2	M. M.	22	F.	Appendix	Muscle	—	Acute appendicitis
3	M. A.	25	M.	Appendix	Submucosa	—	Acute appendicitis
4	J. T.	71	M.	Appendix	Meso-appendix	—	(Post mortem)
5	E. W.	39	F.	Appendix	Muscle	—	Acute appendicitis
6	A. G.	21	M.	Appendix	Muscle	—	Acute appendicitis
7	J. S.	20	M.	Appendix	Subserosa	—	Appendicular colic
8	M. T.	39	F.	Appendix	Subserosa	—	Not known
9	P. C.	33	F.	Appendix	Subserosa	—	Appendicular colic
10	J. S.	13	F.	Appendix	Subserosa	—	Acute appendicitis
11	D. J.	19	F.	Appendix	Subserosa	—	Acute appendicitis
12	P. H.	41	M.	Appendix	Submucosa	—	(Post mortem)
13	A. N.	22	M.	Appendix	Subserosa	—	Acute appendicitis
14	F. C.	9	F.	Appendix	Muscle	—	Acute appendicitis
15	M. W.	49	F.	Appendix	Subserosa	—	(Gynæcological operation
16	D. J.	24	F.	Appendix	Subserosa	—	Appendicular colic
17	J. M.	50	F.	Appendix	Subserosa	—	(Cholecystectomy)
18	M. N.	35	F.	Ileum	Subserosa	—	None
19	W. M.	32	M.	Ileum	Subserosa	—	Acute intestinal obstruction
20	M. C.	70	F.	Ileum	Subserosa	Lymph-node	Chronic intestinal obstruction
21	E. G.	55	F.	Ileum	Mesentery	Lymph-nodes	Chronic intestinal obstruction
22	I. P.	61	F.	Ileum	—	Omentum ? Lymph-nodes	Chronic intestinal obstruction
23	C. B.	55	F.	Ileum	—	Liver Lymph-nodes	Chronic intestinal obstruction
24	J. M.	74	M.	Colon	Pericolic fat	—	Chronic intestinal obstruction
25	I. M.	51	F.	Stomach	Submucosa	—	None

Fifteen of the specimens were obtained at operation and 2 at autopsy. In 8 instances the appendix was acutely inflamed, in 3 operation was performed for appendicular colic, and in 2 the appendix was removed incidentally. In 2 the clinical history is not known.

The largest tumour was 1 cm. in diameter, and none was found in the proximal half of the appendix. Three tumours were confined to the submucosa, in 5 the muscle coats were invaded, and in 8 groups of tumour cells were found in the subserous layer. In 1 case infiltration of the meso-appendix was noted. No remote metastases occurred.

The extra-appendicular cases are recorded in more detail.

Case 18.—M. N., female, aged 35.

History.—In 1930, at the age of 20, this patient underwent laparotomy for a mass in the right iliac fossa, associated with obstructive symptoms. A growth arising in the cæcum and invading the posterior abdominal wall was found, and resection of the right colon with ileo-transverse colostomy was carried out in two stages.

Excision of the tumour posteriorly was incomplete, and post-operative radiotherapy was employed. The pathological diagnosis was fibrosarcoma of the cæcum.

On Oct. 3, 1945 the patient, now aged 35, was operated on by Mr. Stanley Wilson for chronic obstructive symptoms. The ileum one foot proximal to the anastomosis was bound down to an ill-defined mass in the right iliac fossa—presumably a local recurrence of fibrosarcoma. In addition, numerous discrete nodules ½ to 1 cm. in diameter were found in the wall of the small bowel: one was excised for microscopical examination.

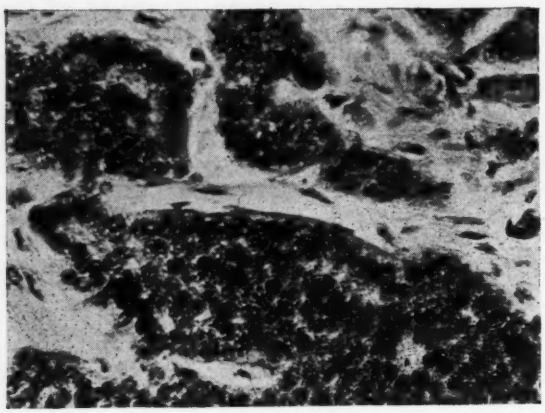

FIG. 181.—*Case* 18. Argentaffinoma of the ileum, showing typical masses of tumour cells, with a tendency to peripheral palisading. (× 180.)

Lateral anastomosis was performed to short-circuit the point of obstruction, and the patient made an uneventful recovery.

Two years later (October, 1947) she still has a palpable mass in the right iliac fossa: it is not increasing in size, and the patient leads a normal healthy life, with no loss of weight.

PATHOLOGY.—The excised nodule consists of yellow tissue, firm in consistence, and 1 cm. in diameter. Microscopically it comprises masses of typical argentaffin cells (*Fig.* 181) which spread diffusely through the submucosa and infiltrate the muscle layers of the bowel. A few groups of tumour cells lie in the subserous layer. The mucosa is intact. The tumour cells are arranged mainly in compact masses, but well-marked rosettes and pseudo-acini are present. The stroma is abundant and fibromuscular in character.

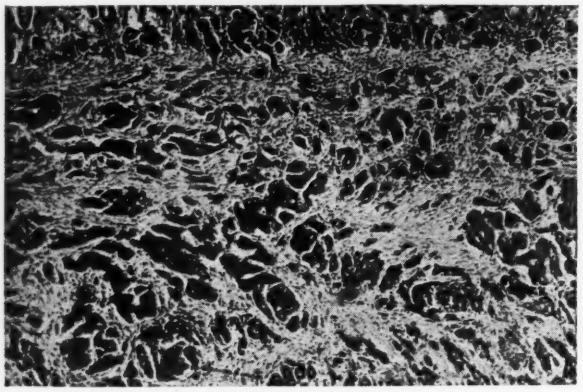

FIG. 182.—*Case* 19. Argentaffinoma of the ileum. Normal mucosa lies along the top of the photograph, and the submucosa is infiltrated by tumour cells. (× 30.)

Comment.—Apart from its very considerable interest as an example of fibrosarcoma of the cæcum, this case illustrates multiple, asymptomatic argentaffinoma of the small intestine, discovered accidentally. The growth infiltrates the bowel wall freely, but does not extend beyond it. One wonders what the ultimate fate of this patient will be, harbouring as she does both recurrent sarcoma and multiple argentaffinoma.

Case 19.—W. M., male, aged 32.

HISTORY.—The patient's first symptoms were severe colicky abdominal pain and vomiting when he was admitted to Dunedin Hospital on June 25, 1946.

ON EXAMINATION.—The abdomen revealed neither distension, visible peristalsis, nor a palpable mass. There was slight tenderness in the right iliac fossa, but no guarding. Loud borborygmi were heard on auscultation.

AT OPERATION (Mr. Norman Speight).—Lower paramedian incision. The peritoneum contained free fluid. Collapsed small bowel was followed from the ileocæcal junction upwards for several feet to a point of obstruction. Here the intestine was acutely kinked, at the site of a small palpable tumour. This was removed by resection of a wedge-shaped portion of bowel wall, which was repaired transversely by two layers of sutures, the lumen being left quite patent. Post-operative progress was uneventful.

PATHOLOGY.—The specimen (*Fig.* 181) consists of a segment of bowel wall, invaginated by a tumour mass 1 cm. in diameter. This is firm, yellow, superficially ulcerated, and lies mainly in the submucous coat. The cut surface reveals an intact muscle coat, which is acutely angulated or 'buckled'.

Sections show groups of polygonal cells with large vesicular nuclei, lying in a moderately dense fibrous stroma (*Figs.* 182, 183). The cell groups are small, and some form pseudo-acini, while others show palisading of the peripheral cells. Groups of cells extend through the muscle coats to the subserosa. There is moderate infiltration by inflammatory cells of both large and small mononuclear types.

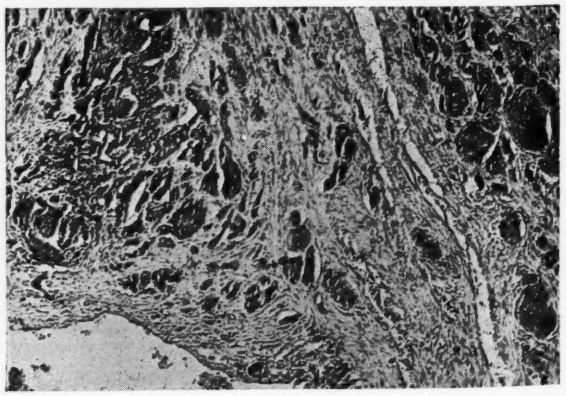

FIG. 183.—*Case* 19. Showing tumour cells extending into the subserous layer. (× 30.)

Comment.—This case demonstrates acute intestinal obstruction resulting from a small argentaffinoma of the ileum. The tumour is single and has not metastasized. The mechanism whereby obstruction was caused was acute angulation of the bowel.

Case 20.—M. C., female, aged 70.

HISTORY.—The patient complained of recurrent episodes of colicky epigastric pain, dating from 1933. In 1935 the gall-bladder had been removed, with little relief. On Jan. 28, 1937 the patient was admitted to

Oamaru Hospital, complaining of intermittent pain, vomiting, and constipation. There was no diarrhœa, dyspepsia, or loss of weight.

On Examination.—Negative, except for the presence of a tender mobile mass in the right lower abdomen.

At Operation.—The condition found was one of chronic small-bowel obstruction, with distension and hypertrophy of the intestine down to an annular growth in the terminal ileum. In addition, coils of bowel were adherent to a large single mesenteric gland, 4 cm. in diameter. Resection of the primary tumour along with the gland-bearing portion of the mesentery was effected

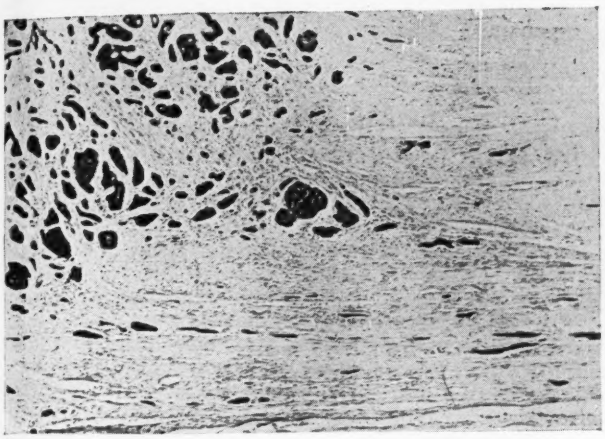

Fig. 184.—*Case* 20. Argentaffinoma of the ileum, showing typical cell masses and cords extending through the muscle layers. (× 30.)

with difficulty. The patient died a few hours after operation.

Pathology.—The portion of excised bowel contains a small tumour growing halfway round its circumference. There is invagination of the muscle wall, with resultnig kinking. The growth is hard, and greyish-white in colour. Microscopically (*Fig.* 184) it consists of compact masses of typical polygonal argentaffin cells. These infiltrate from the submucous to the subserous layers, the mucosa being intact. The gland is largely replaced by cells of characteristic argentaffin appearance and arrangement (*Fig.* 185).

Comment.—This is a case of single argentaffin tumour of the ileum, which had caused intermittent obstructive symptoms for four years, and had metastasized to a mesenteric lymph-node.

Case 21.—E. G., female, aged 55.

History.—The patient was admitted to Dunedin Hospital on Sept. 22, 1939, complaining of dyspepsia and loss of weight of nine months' duration. For the last four months she had been having intermittent attacks of colicky central pain and vomiting. Constipation alternated with diarrhœa, and flatulence occurred, particularly just after meals. The patient had been conscious of a lump in the abdomen for two months.

On Examination.—A spare middle-aged female who had obviously lost weight. A large, firm mobile mass was present in the right upper abdomen. Over this there was slight tenderness but no rigidity. The provisional diagnosis was one of carcinoma of the hepatic flexure of the colon, but barium enema and X-ray examination revealed no lesion.

At Operation (Prof. Gordon Bell).—The abdomen was explored through a right upper paramedian incision. The mass was found to consist of matted coils of small bowel, adherent to tumour tissue which was invading the mesentery. A hard gland, 4 cm. by 3 cm. in size, lay at the root of the mesentery, and was adherent to the superior mesenteric artery; 6 feet of matted bowel and the associated tumour-containing mesentery was resected, and the bowel reconstituted by end-to-end anastomosis. It was not possible to remove the mesenteric gland on account of its adhesion to the superior mesenteric artery; it was almost certainly a secondary malignant growth. (Cf. *Case* 20.)

Progress.—The patient made a good recovery, and eight years later (October, 1947) is in good health. She had regained her normal weight of 10 st. 3 lb. There is a small tender palpable lump just below and to the right of the umbilicus—this is probably the gland

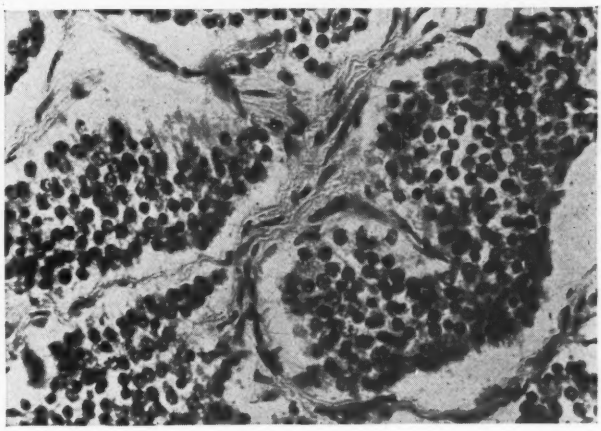

Fig. 185.—*Case* 20. Section of mesenteric lymph-node showing secondary argentaffinoma. (× 180.)

observed at operation, and it appears to be non-progressive.

Pathology.—There appear to be two primary tumours, infiltrating and coalescing in the wall of the bowel, and extending into the mesentery. The mucosa is intact. In the mesentery lies a hard, fibrous mass,

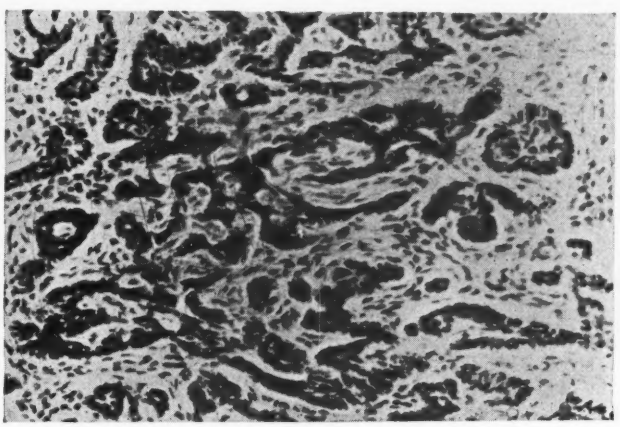

Fig. 186.—*Case* 21. Argentaffinoma of the ileum, showing masses of tumour cells and well-marked peripheral palisading. (× 90.)

which measures 6 cm. by 4 cm. and is greyish-white in colour. Microscopically the appearances are typical of argentaffinoma (*Fig.* 186).

Comment.—This is a case of multiple argentaffinoma of the ileum, which resulted in chronic intestinal obstruction. Metastasis had taken place to the mesentery, and almost certainly to the lymph-gland, which was enlarged, but which has been present now for eight years without progression.

Case 22.—I. P., female, aged 61.

HISTORY.—Admitted to Auckland Hospital on Dec. 3, 1939.

The patient had enjoyed good health up to seven months prior to admission. During this period she had suffered from constipation, abdominal pain and vomiting, and had lost 2 st. in weight. She had had two attacks of diarrhœa, each lasting several days, without blood in the stools. The patient had noticed an abdominal swelling,

FIG. 187.—*Case* 22. Section of nodule from omentum —secondary argentaffinoma. (× 180).

which was intermittent; its appearance coincided with pain, and its disappearance with relief and release of flatus.

ON EXAMINATION.—Physical examination was negative except for the presence of a mobile abdominal swelling lying transversely below the umbilicus. This was tympanitic, and could be made to disappear by firm pressure. X-ray examination revealed a distended coil of small bowel.

AT OPERATION (Mr. K. MacKenzie).—The small bowel was found to be distended and hypertrophied above a ring stricture of the terminal ileum. At this point there was a palpable tumour, and the bowel was adherent to a mass of enlarged lymph-glands which lay partly in the root of the mesentery, and extended upwards along the aorta. The omentum was studded with multiple carcinomatous nodules. Lateral entero-anastomosis was carried out to short-circuit the obstruction, and biopsy material was taken from the mesenteric glands and omentum. The patient made a good post-operative recovery.

PATHOLOGY.—Sections of omentum (*Fig.* 187) show masses of typical argentaffin cells, in which argyrophil granules are demonstrated by means of Masson's silver stain. The lymph-node shows chronic inflammatory changes, but no evidence of metastasis.

Comment.—In this case a constricting argentaffinoma of the ileum was the cause of chronic intestinal obstruction, and was associated with widespread peritoneal carcinomatosis. It has unfortunately not been possible to trace this patient, but presumably she has died.

Case 23.—C. B., female, aged 54.

HISTORY.—Admitted to Dunedin Hospital on Dec. 7, 1936, at the age of 54, complaining of attacks of colicky pain which dated from 1932. The present attack had lasted for seven days: the pain was umbilical, and was associated with vomiting and diarrhœa. The patient gave a history of flatulent dyspepsia with intolerance to fatty foods.

Examination revealed only slight tenderness in the right upper quadrant. The provisional diagnosis of biliary colic was made, and operation recommended. The gall-bladder and appendix were removed, but were apparently normal. The liver was greyish, and was studded with small brown nodules, one of which was removed. Exploration of the abdomen failed to reveal the site of the primary tumour.

Pathological report on the specimen from the liver diagnosed secondary carcinoma of solid type, but did not specify the type or probable source. Re-examination of the sections (*Fig.* 188) shows that the cellular deposits are typical of argentaffinoma. Tumour cells arranged compactly in well-defined masses lie chiefly in the periportal tissues. The cells are regular in shape and size, and possess large round vesicular nuclei, with sharply defined nuclear membranes. The cytoplasm is moderate in amount, and is finely granular.

On Jan. 9, 1938, the patient was readmitted, with the history that she had continued to have attacks of pain, vomiting, and diarrhœa, first at three-monthly and now at weekly intervals. She still complained of flatulent dyspepsia. There was no loss of weight, and the liver was not palpably enlarged. The patient was discharged without further treatment.

On July 18, 1939, she returned complaining of almost constant pain. There was still no weight loss, no enlargement of the liver, no abdominal distension, and no free peritoneal fluid apparent on clinical examination.

On Feb. 26, 1940, the patient reappeared. In addition to the previous symptoms she had now lost weight, and was too weak to do her housework. Watery diarrhœa had become a major complaint. Laparotomy was

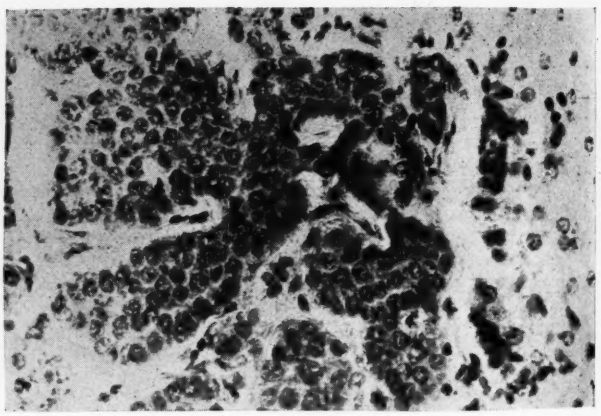

FIG. 188.—*Case* 23. Secondary argentaffinoma of the liver. (× 180.)

performed (Mr. Gordon Bell). Free peritoneal fluid of opalescent appearance was found, and the small bowel was revealed to be the seat of a growth which produced annular constriction. Above this the bowel was distended and hypertrophied. The mesentery contained numerous hard enlarged glands, and the liver, which was not seen, was felt to be shrunken and irregular. The point of obstruction was short-circuited by entero-anastomosis and the patient made a good post-operative recovery.

In January, 1941, the patient returned to hospital in an advanced condition of cachexia, and complained of pain, vomiting, and melæna. The abdomen revealed distension, with visible peristalsis, gross enlargement of the liver, and an ill-defined large hard mass in the region of the umbilicus. The patient died, but unfortunately a post-mortem was not obtained.

Comment.—In this interesting case, symptoms had been present for nine years prior to death. The initial attacks of colic were diagnosed as biliary in origin, and at laparotomy secondary carcinoma of the liver was discovered. Since the primary was not found at this time, it may be presumed to have been very small. Loss of weight was delayed until eight years after the commencement of symptoms, and four years after the discovery of hepatic metastases. Failure of recognition of the true nature of these secondary growths delayed further operative intervention, which might well have prolonged considerably the life of the patient.

Case 24.—J. M., male, aged 74.

HISTORY.—Admitted Dunedin Hospital on Dec. 30, 1946.

The patient complained of lower abdominal pain, flatulence, and constipation of eight months' duration, with nausea and vomiting during the last six weeks. There had been no diarrhœa, no loss of weight, and no blood in the stools.

ON EXAMINATION.—A plump elderly man of healthy appearance. The abdomen was distended, chiefly in the right flank and epigastrium. No mass could be felt, and rectal palpation was negative. Barium enema showed a stricture extending over 2½ in. of the middle of the descending colon, the appearance being that of annular carcinoma.

OPERATION (Mr. Geoffrey Barnett).—This took the form of a Paul-Mikulicz resection of the descending colon, after mobilization. No secondary spread of the tumour was found in liver, lymph-glands, or peritoneum. The patient made a good post-operative recovery, and the colostomy was duly closed.

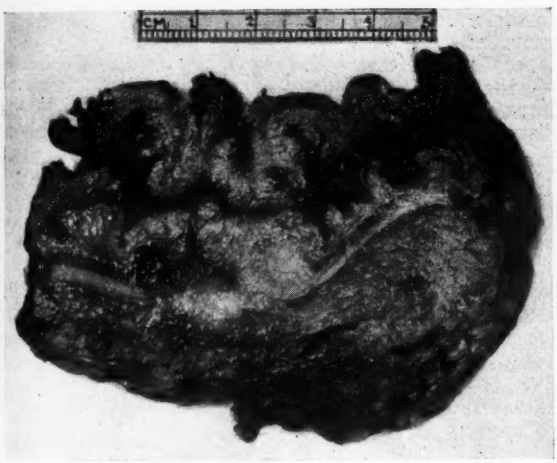

FIG. 189.—*Case* 24. Gross specimen, argentaffinoma of the colon, completely interrupting the muscle coats and extending into the pericolic fat.

PATHOLOGY.—The specimen consists of a portion of descending colon, containing a tumour which completely encircles the bowel over a distance of 2½ in. The tumour tissue is greyish-yellow in colour. On section it is seen to be invading the muscle coat, which is completely interrupted, and extending into the pericolic fatty tissue (*Fig.* 189).

Microscopical examination shows large sheets of cells infiltrating the submucous and muscle layers, which are broken up and destroyed. The cells are moderately hyperchromatic, but are regular in size and shape (*Fig.* 190). There is no attempt at acinus formation. The mucosa is substantially intact, and is composed of normal epithelium.

Comment.—This tumour is a characteristic argentaffinoma, occurring in a very rare site in the descending colon. A year later this patient is alive and well, with no sign of recurrence.

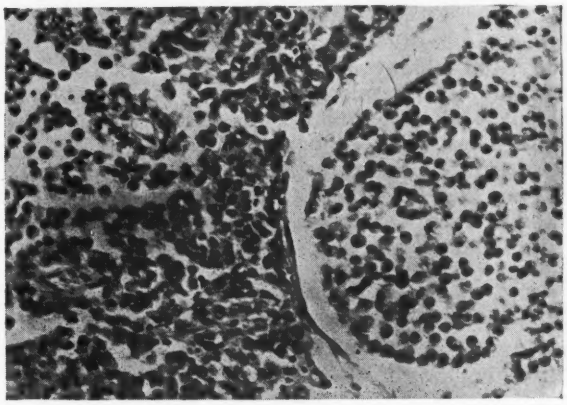

FIG. 190.—*Case* 24. Argentaffinoma of the colon, composed of large sheets of carcinoid cells. (× 90.)

Case 25.—I. M., female, aged 51.

HISTORY.—Admitted Dunedin Hospital on March 26, 1947 and died two days later. At post mortem the cause of death was found to be dissecting aneurysm of the aorta with hæmopericardium. A small tumour was found in the stomach, 0·5 cm. in diameter; it was of yellow colour and was limited to the submucosa.

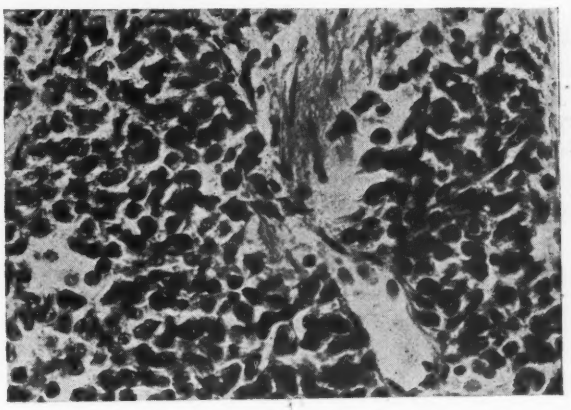

FIG. 191.—*Case* 25. Argentaffinoma of the stomach. The tumour consists of large sheets of rather hyperchromatic cells. (× 180.)

Microscopical examination revealed compact masses of regular cells of argentaffin type (*Fig.* 191).

Comment.—This is an asymptomatic, solitary argentaffinoma of the stomach.

DISCUSSION

The 6 cases of argentaffin tumour of the small intestine reported above comprise 5 females and 1 male. This predilection for the female sex has not generally been found in connexion with extra-appendicular carcinoids.

The average age of the patients is 51·1 years. It is of considerable interest that the 2 tumours which occurred in patients below the age of 40 were benign, while the other 4, occurring in patients over 50, were all associated with secondary spread.

Symptoms due to the growth were present in 5 cases and absent in 1. One presented as acute intestinal obstruction without antecedent history. The remaining 4 patients complained of symptoms, the duration of which ranged from six months to eight years. The clinical picture was that of subacute intermittent small-bowel obstruction, and comprised chiefly recurrent colicky central pain, vomiting, and flatulent dyspepsia. In 2 patients a diagnosis of cholelithiasis was made, and the gall-bladder was removed without benefit.

Significant loss of weight occurred in 3 patients, and in 4 a palpable abdominal mass was noted. Bleeding from the bowel occurred in only 1 case as a terminal event, but 3 patients complained of recurrent diarrhœa.

The growth was single in 4 and multiple in 2 instances. Secondary spread had taken place in 4, consisting of lymph-node enlargement in 3, local infiltration of the mesentery in 1, hepatic deposits in 1, and diffuse peritoneal carcinomatosis in 1 case.

The cause of obstruction was ' buckling ' of the bowel wall in 2 instances, annular constriction in 2, and infiltration of the mesentery with adhesion of matted coils of bowel in 1.

The 2 patients in whom no metastasis was found are alive and well, two years and sixteen months after operation respectively. Of the 4 malignant cases 1 died after operation, and 1 died nine years after the commencement of symptoms. One patient is alive and well eight years after operation, at which a tumour involving bowel and mesentery was resected but a large gland left in situ. One case is untraced.

Cases 24 and 25 are of interest in view of the rarity with which the colon and stomach are subject to argentaffinoma.

TREATMENT

The treatment of benign argentaffinomas requires no special consideration, local excision being adequate. As regards the malignant tumours, the extremely slow growth of both primary and secondaries justifies strenuous efforts at complete extirpation yet at the same time condones, in poor-risk patients, measures which would be regarded as incomplete and ill-advised in the treatment of adenocarcinoma.

Thus, excision of the primary tumour is not necessarily contra-indicated by the presence of lymph-node, peritoneal, or even hepatic metastases. Indeed, in favourable circumstances, every effort should be made to excise metastatic deposits. Stewart and Taylor (1926) record the example of a young woman from whom were removed two large pelvic masses secondary to carcinoid of the appendix ; the patient was alive and in good health ten years later.

On the other hand, Porter and Whelan (1939) cite a case in which a primary argentaffinoma of the ileum was excised in the presence of a large secondary retroperitoneal mass. Twenty years later the patient was still alive, and the mass apparently unchanged.

The susceptibility of the growth to radiotherapy is not known, although Ariel (1939) suggests that its use may be indicated for inoperable masses.

SUMMARY

Twenty-five cases of argentaffin tumour of the gastro-intestinal tract are presented. Of these, 8 are extra-appendicular in site, and they include examples involving the colon and the stomach.

The incidence, pathology, and treatment of the tumour are discussed.

My thanks are due to the Medical Superintendents, surgeons, and pathologists of the hospitals mentioned in the text, for access to and permission to publish this material. For the microphotographs I am indebted to Mr. Howard, of the Department of Anatomy in the University of Otago.

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NEUROGENIC TUMOURS OF THE STOMACH

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BENIGN tumours of the stomach are rare, and reports of those of neurogenic origin have not appeared frequently in the surgical literature. Two cases in which tumours of the stomach of this type occurred are herewith reported.

DEFINITION

Tumours of neurogenic origin form a fairly well-defined group, the pathology of which is complex, but which can be recognized by certain specific histological features. The nomenclature of these tumours is extremely varied and confused, and, judging from the variety of names still employed in reporting them, is by no means yet standardized. There is also still no uniformity of opinion as to their histogenesis.

Virschow in 1862 divided tumours of peripheral nerves into two groups which he called false and true neuromata, the true neuromata arising from nerve-fibres and nerve-cells, and the false from the nerve-sheaths. The modern conception is that the only true neuromata are those arising from the primitive cells of the sympathetic and chromaffin systems, and these are certainly less common than the nerve-sheath tumours.

The false neuromata are now thought by most authorities to arise from the specialized investment surrounding nerves, although Anderson (1920) reported a case of neurofibromatosis with tumours arising from the optic and olfactory nerves, which tends to disprove this view. Verocay (1910), Antoni (1920), Masson (1932), Stout (1940), and others believe that they arise from the sheath of Schwann and are thus neuro-ectodermal in origin; whereas others, foremost amongst whom are Mallory (1920) and Penfield (1937), hold that they arise from modified fibroblasts of the connective-tissue sheath of Henle, or endoneurium, and from the perineurium and are thus mesodermal in origin. Even with special staining methods, and though a considerable amount of experimental work has been done in this field of pathology, there is still no agreement, nor is it possible to be dogmatic as to their exact site of origin. This controversy is well presented in a paper by Bailey and Hermann and the consensus of opinion is that one type of nerve-sheath tumour, the neurilemoma, is composed primarily of Schwann cells, the other group, the neurofibroma, of Schwann cells and connective tissue combined together.

Tumours of neurogenic origin may occur all over the body and especially in the extremities, thorax, abdominal cavity, mesenteries, and retroperitoneal spaces. This paper is only concerned with those occurring in the stomach.

NOMENCLATURE AND CLASSIFICATION

A satisfactory, comprehensive, and simple classification of intra-abdominal neoplasms of neurogenic origin is that employed by Ransom and Kay (1940) :—

I. Nerve-sheath Tumours :—
  A. Benign :
    1. Neurilemoma (schwannoma, perineural fibroblastoma, etc.)
    2. Neurofibroma (of type associated with von Recklinghausen's disease).
    3. Cirsoid or plexiform neurofibroma.
    4. Ganglionated neurofibroma.
  B. Malignant :
    1. Neurogenic sarcoma.

II. Neuroblastic tumours of the sympathetic system :—
    1. Sympatheticoblastoma.
    2. Paraganglioma.
    3. Ganglioneuroma.

The cirsoid or plexiform neurofibroma sometimes known as elephantiasis nervorum does not appear to have been described as occurring in the stomach, although such tumours may occur in the intra-abdominal cavity; whereas the ganglionated neurofibromata are invariably retroperitoneal when they occur in this situation. The true neuroblastic tumours are not included in this account. Thus there are two types of benign nerve-sheath tumours that occur in the stomach :—

1. The neurilemoma, usually solitary and unassociated with von Recklinghausen's disease;
2. The neurofibroma, often multiple and usually associated with von Recklinghausen's disease;

and the malignant tumours of nerve-sheath origin.

This classification is essentially a clinical one and it must be understood that the differentiation, though possible, is not so simple on pathological grounds. These two groups of tumours are closely allied and the distinction is made more difficult by the multiple and varied terminology which has been applied to them.

Verocay (1910), who first called attention to the histology of nerve-sheath tumours, segregating them as a distinct pathological entity, used the name neurinoma to describe them. Other pathologists, according to their interpretation of the histogenesis,

have called them perineurial fibromata or fibroblastomata (Penfield, 1937); schwannomata (Masson, 1932); lemmomata, schwannoblastomata, peripheral gliomata, and neurilemomata (Stout, 1935). The lastnamed, who has made valuable contributions to the literature on the subject of nerve-sheath tumours, points out that the name neurinoma, inferring nerve-fibre tumour, is an incorrect one, and coined the name neurilemoma or nerve-sheath tumour and supported their nerve-sheath or Schwannian origin. This name has been more generally adopted than any other, though often spelled incorrectly with two m's, and will be used in this paper to describe tumours of the first group.

The term neurofibroma has often been loosely applied to describe all tumours of nerve-sheath origin (Cutler and Gross, 1936, b), and the discussion is complicated by the fact that tumours of both types may be associated with von Recklinghausen's disease—the solitary neurilemoma of the stomach, from a review of the literature, apparently rarely; the neurofibroma almost invariably—and thus forming clinically a fairly well-defined group, although they too may occur as solitary tumours. If we are to retain the term neurofibromatosis as synonymous with von Recklinghausen's disease, it seems reasonable to describe those tumours which are in fact commonly associated with this disease as neurofibromata.

Both types of tumour exhibit rather similar and typical tissue on histological examination, to be described later; but the pathological characteristics of neurofibromata are not so clearly defined or easily recognizable, for whereas the neurilemoma is thought to consist largely of well-differentiated cells derived from Schwann cells, neurofibromata are composed of Schwann cells and fibrous tissue combined in varying proportions.

In von Recklinghausen's disease, or hereditary generalized neurofibromatosis, multiple tumours of the latter type are present, and the nearer the periphery they occur the more the fibrous tissue element predominates, the tumours showing less of the characteristics of neurilemoma and more those of the fibroma. It has been suggested that all nerve-sheath tumours are merely local manifestations of von Recklinghausen's disease: neurilemomata of the stomach do appear, however, to occur as solitary tumours with far greater frequency and to form a separate clinical group. Von Recklinghausen's disease was associated with 18 per cent of 50 cases of neurilemoma in other situations quoted by Stout, and it is thus impossible to deny that a solitary tumour does not imply the presence of systemic disease. It is, however, difficult to determine with accuracy how often this association does in fact occur, for, as Stout (1935, b) rightly points out, the stigmata of von Recklinghausen's disease may not easily be recognized or even looked for unless the significance of the primary tumour is realized. From a review of the literature the association is unusual. None of the 7 cases of neurogenic tumours of the stomach reported by Ransom and Kay (1940) were associated with von Recklinghausen's disease, nor any of the cases reported by Geschickter (1935, a). Likewise it is difficult to assess how often, if ever, a true solitary neurofibroma occurs in the stomach unassociated with von Recklinghausen's disease, though it is agreed that a neurofibroma does sometimes occur as a solitary tumour. This appears to be very rare, if they occur at all, for single benign neurogenic tumours of the stomach are often reported in the literature as neurofibromata when they conform in fact clinically and pathologically to the neurilemoma group.

Geschickter (1935, a), in describing a clinico-pathological classification of nerve-sheath tumours, suggests that the fundamental difference in the histogenesis of the two groups may be due to the presence or absence of a myelin sheath of the nerve affected; the neurilemomata arising from medullated nerves, the fibrous reaction of the perineurium accounting for the typical encapsulation of tumours of this type which is absent in neurofibromata which tend to arise from the non-myelinated fibres of the visceral sympathetic nerves and the nerves of the skin at their terminations.

When there is a more thorough general knowledge of the histogenesis of nerve-sheath tumours and a clearer understanding of their pathology, a more satisfactory classification may be established. At present it is somewhat confused and there is no clear distinction between the two types. The association of the two types with von Recklinghausen's disease may be summarized thus:—

1. Solitary neurilemoma—relatively common.
2. Neurofibroma associated with von Recklinghausen's disease—relatively common.
3. Neurilemoma associated with von Recklinghausen's disease—rare.
4. Solitary neurofibroma—rare.

INCIDENCE

Since Verocay (1910) first called attention to the histology of these tumours, a number of cases have been reported in the literature, although the number is difficult to assess owing to the many undoubted misinterpretations of the pathology and the confusion in the nomenclature. However, some idea of their frequency can be judged from the figures quoted below.

Rigler and Ericksen (1936) estimate the frequency of benign tumours of the stomach of all types as 26 per cent of all gastric neoplasms in 6742 autopsies performed. Eusterman and Senty (1922) reported 2168 neoplasms of the stomach, of which 27 were benign tumours, an incidence of 3 per cent, but reported no neurogenic tumours; Minnes and Geschickter (1926) 50 cases of benign tumours of the stomach, of which 1 was a neurinoma. They mention 102 cases of neurogenic tumour among 931 benign tumours collected from the literature, or 10·9 per cent of recorded gastric tumours. Balfour and Henderson (1927), in reporting 58 benign tumours, found no neurogenic tumours. Eliason and Wright (1925) also found no neurogenic tumours amongst 50 benign tumours of the stomach. Geschickter (1935, b) found 10 nerve-sheath tumours among 962 malignant lesions of the digestive tract, 3 of these occurring in the stomach. Shallow and Lemmon (1941) reported 13 benign tumours of the stomach in 300,000 cases, with 1 neurofibroma; Mason and Dwyer (1928, a) 3 cases of benign

tumour in 4000 examined by X rays and clinically, but no neurogenic tumours. Root (1942) reported 17 benign gastric tumours in 250,000 cases, amongst which there were 3 neurogenic tumours and 3 leiomyomata. Ransom and Kay (1940), in a paper on this subject, describe 18 cases of neurogenic tumours occurring in the abdomen, 7 in the stomach; and Stout (1935, b) collected 35 tumours of this type from the literature, adding 3 cases of his own. In recent years several other small series and individual cases have been reported.

No attempt has been made to collect and classify all these cases owing to the confusion in the nomenclature and the lack of histological evidence of many of the cases reported.

Of all benign tumours of the stomach, neurogenic types are probably not the rarest. Benign tumours of all types are probably commoner than is usually imagined, for, as Lockwood (1943) points out, they are more often discovered post mortem than on the operating table. He considers the incidence of benign tumours of all types to be 4·5 per cent (12 cases amongst 266 malignant lesions in his series).

Mallory (1920), when discussing neurogenic tumours in this situation, stated that he considered them to be more common than leiomyomata. The latter are certainly among the commoner of benign tumours—35 per cent of all benign tumours of the stomach (Lahey and Colcock, 1940); 39 per cent (Collins and Collins, 1938). Mallory also stresses the difficulty in differentiating the two pathologically without special staining methods. Stout (1941) stresses the same point in a further paper concerning the leiomyomata, and reclassifies as leiomyomata 3 cases which he originally classified as neurilemomata. This differentiation will be considered further when discussing the pathology. As leiomyomata of the stomach are clinically, radiologically, and on macroscopical examination precisely similar to solitary nerve-sheath tumours, a certain number of cases of tumours of both types have probably been wrongly interpreted at microscopy. Most, if not all, of the 42 cases reported by Gosset (1924) as gastric schwannomata are thought to be of myogenic origin. It may well be that benign nerve-sheath tumours of the stomach, though rare, are commoner than is generally imagined. At present few surgical text-books mention their occurrence in this site, though leiomyomata are almost invariably described.

AETIOLOGY

Masson (1932) has attempted to show a relationship between proliferation of the nervous tissue in the stomach wall associated with inflammation of the gastric mucosa and with gastric ulcers, and has studied similar changes in the walls of obliterated appendices from this point of view. Askanazy (1926) also investigated the local changes in the nerves in association with gastric ulcer and quoted a case of Dupuy (1925) in which a diffuse neuroma occurred in the stomach wall of a patient following attempted suicide by the ingestion of hydrochloric acid, the stomach wall being subsequently examined at post-mortem. There is little evidence that there is any relationship between these nervous proliferations and true benign neurogenic growths, and the aetiology and predisposing factors are quite obscure.

The two types of tumour will now be considered in more detail.

NEURILEMOMA

As well as occurring in the stomach and retroperitoneal regions, these tumours also occur in the limbs, especially the upper, in the thorax, neck, and trunk. As already stated, they are not necessarily solitary neoplasms and may be, though rarely, associated with von Recklinghausen's disease. They probably arise from the sheath of the sympathetic fibres of the submucosal and myenteric plexus of Auerbach of the small intestine. Most tumours appear to originate in the latter, according to Schrager (1939), which is situated between the annular and longitudinal muscle coats. Their actual connexion to nerves can seldom be demonstrated on histological examination.

Pathology.—Full accounts of the pathology of neurilemomata are to be found in the writings of Verocay (1910), Masson (1932), Antoni (1920), Mallory (1920), Penfield (1937), Stout (1935, b), and others, and Foot (1940) has made a very comprehensive review of the subject.

The pathology is somewhat complex and is not considered in detail in this paper. The tumour is composed primarily of Schwann cells, and the very characteristic feature on examination is the so-called palisade arrangement of the elongated nuclei, connected in a syncitium which is nearly always present and betrays their neurogenic origin. Two types of cell are usually described as composing the tumour, the so-called Antoni type A and Antoni type B occurring in varying proportions.

Type A consists of large, slender, fasciculated, elongated cells arranged in palisades, the ends of the cells being rounded and giving rise to long fine processes which anastomose with similar processes from other cells to form a syncitium. These end-processes have a thin covering of collagenous material and large wavy collagen fibres are scattered throughout the tumour. The nuclei are hyperchromatic, thin and elongated in shape, and arranged parallel to each other in palisades like ' staves of a barrel '—Verocay (1910). They also occur in whorls and interlacing patterns. Masson (1932) ascribes the palisading effect to proliferation of the nuclei having occurred within the endoneurium.

The Antoni type B cells are composed of a reticular type of tissue of myxomatous character which is undifferentiated and can be best demonstrated with silver stains. These cells are irregularly scattered, with intercellular fluid and minute cystic spaces present, and hyaline degeneration is present in the blood-vessel walls. Masson considers that this type of tissue results from degenerative changes.

The neurilemoma is encapsulated and poorly vascularized and liable to secondary changes and necrosis. The characteristic niche of the mucous surface probably develops from a central necrotic focus which gradually spreads to the mucosa and perforates through to the gastric lumen, being later enlarged through the action of gastric juice on its edges, giving rise to the typical punched-out appearance (*see Fig.* 193). In leiomyomata and other

benign tumours, a similar central ulceration occurs and is probably then due purely to mechanical causes.

Mallory (1920) and Stout (1935, b) have emphasized the fact that neurilemomata are easily confused histologically with leiomyomata, and according to Foot (1940), various writers, notably Gosset (1924), have fallen into the error of considering a palisaded nuclear architecture as being a specific feature of nerve-tissue tumours. This formation is also seen in leiomyomata and sometimes in sarcomata. The pedunculated tumours of the stomach described by Gosset resemble muscular neoplasms rather than neurilemomata. Leiomyomata also have a tendency to undergo patchy necrosis simulating the Antoni type B tissue of neurilemomata, making the differentiation still less easy. Stout finds the absence of encapsulation in smooth muscle tumours to be characteristic and this should be helpful in differentiating the two types, the similarity being in fact a relatively superficial one. Neurilemomata are mostly benign tumours, but may assume malignant characteristics at any time.

NEUROFIBROMA

As already stated, this group of tumours is closely related to the above, but has not so clearly defined or easily recognizable characteristics and they are thought to be composed of Schwann cells and fibrous tissue in varying proportions, depending largely on their anatomical situation. They are associated always or almost always with von Recklinghausen's disease, any of the characteristic stigmata of which—cutaneous nodules, pigmented areas in the skin, mental deficiency, and skeletal changes—may be present in an individual case. They tend to be multiple and many cases of so-called neurofibromata described, unassociated with von Recklinghausen's disease, should probably not be classed as such. Apart from the usual association with von Recklinghausen's disease, the distinction between the two groups of tumour is entirely a histological one, depending largely on a relative preponderance of fibrous tissue. The distinction, however, is not entirely academic, due to the marked difference in liability to malignant change in the two types.

Schwartzman, Grossman, and Dragutsky (1943) stress the importance of bearing in mind the visceral manifestations of von Recklinghausen's disease when this disease is present, and the symptoms suggest that they occur in the cranial, spinal, thoracic, and bony structures as well as in the abdominal cavity, and describe a case of an abdominal mass occurring in a patient with generalized neurofibromatosis. Grill and Kuzma (1942) have reviewed the cases of generalized neurofibromatosis occurring in the intestine, but do not include any cases of gastric origin.

Pathology.—The same elongated spindle cells are present, but there is no characteristic arrangement as in the neurilemoma, the cells being arranged in interlacing strands with varying amounts of collagenous and myxomatous intercellular substance. They are composed largely of fibrous and fibroblastic tissue of loose, delicate, reticular type in interlacing strands corresponding to Antoni type B tissue. Typical palisading is absent (*Fig.* 197). The more peripherally situated tumours of von Recklinghausen's disease contain an increased amount of connective tissue, and melanin may be present. The tumours of this type are not encapsulated and they have a not inconsiderable tendency to malignant change. This particularly applies to the more deeply placed tumours.

MALIGNANT TUMOURS OF NERVE-SHEATH ORIGIN

Sarcomatous changes in neurilemomata giving rise to metastases have frequently been reported. The differentiation between benign and malignant tumours may not be easy and depends largely on the cellularity of the tumour and the presence of infiltration of the stomach wall. Malignant change had occurred in 3 of the 8 cases reported by Ransom and Kay (1940). Distant metastases do not occur early in the disease, but malignant degeneration may occur at any time. Malignant change has also occurred at a later date in cases where neurilemomata have been incompletely removed. Generally they tend to remain locally malignant for long periods and metastasize late, and the prognosis in the malignant type is thus good. Harrington (1934) emphasizes the same point in reporting 14 cases of intrathoracic neurilemomata and advises surgical removal in all cases, as malignant change is liable to take place at any time. Neurilemomosarcoma would seem to be a suitable though cumbersome name for this type of neoplasm.

The encapsulated neurofibromata have an even greater tendency to malignant change to form spindle-celled fibrosarcomata or neurofibrosarcomata. This type may also occur *de novo* (Stewart and Copeland, 1931). Hosoi (1931) states the incidence of malignant change to be 13 per cent—16 out of 65 cases studied. Trueblood (1941) states 10 to 15 per cent, and Lewis and Hart (1930), in their own series, find an incidence of 50 per cent, though they think 12 per cent a more likely figure. Stout (1935, a) finds the incidence of metastases to be 22 per cent. All writers find the tendency to malignant change greater than in neurilemomata. The malignant tumours, when they occur, consist of spindle cells in interlacing bundles and are characterized by their persistent growth, the presence of metastases, and their reappearance after excision.

GROSS CHARACTERISTICS OF BENIGN NERVE-SHEATH TUMOURS OF THE STOMACH

The tumours are usually small and less than 6 cm. in diameter, solid, and sometimes lobulated and attached to the stomach wall by a broad base but they may be pedunculated. They are greyish or pink in colour and usually single. The mucous membrane covering the tumour is vascular and often ulcerated; a very characteristic feature is the deep punched-out ulceration on the surface of the tumour which accounts for the characteristic niche often seen on X-ray examination.

There is no characteristic site of origin of these tumours in the stomach. Stout states that they are more often encountered on the anterior surface

of the stomach, where they are also more accessible to clinical examination. Nervous tissue is more abundant in the lesser curve, which he thinks accounts for their frequent occurrence in this situation. Ransom and Kay (1940) also favour the lesser curve as the most frequent site, while Senturia (1940) thinks the posterior wall is a common site, this accounting for the excessive hæmorrhage often associated with them. Odquist (1937) favours the greater curvature, and several have been reported where the tumour has prolapsed through the pylorus, causing obstructive symptoms. There thus appears to be no characteristic site and they may occur in any part of the stomach wall.

They may be submucous, subserous, or 'hour-glass' in position, more frequently the former, according to their relationship to the gastric wall. This differentiation has an important bearing on the symptomatology. On gross examination there are no distinguishing characteristics to differentiate these tumours from leiomyomata.

Clinical Features.—There are no characteristic clinical features which distinguish these tumours. The symptoms will depend to a certain extent on their position and situation in the stomach wall. Hæmorrhage is the most common symptom (in 50 per cent, Odquist, 1937); probably around 80 per cent of cases exhibit this symptom. Hæmorrhage is usually considerable and occurs at intervals, the patient being well in the intervening periods but with a steadily progressive anæmia. Hæmorrhage may occur in the form of hæmatemesis and less frequently melæna and only occurs in the presence of ulceration. The tumour, when situated near the pylorus, as with other benign tumours, tends to become pedunculated and to prolapse through the lumen, giving rise to recurrent abdominal pain and vomiting. Occasionally pain may simulate peptic ulceration (Senturia, 1940) which is in fact present. In the subserous type symptoms may be absent and a mass may only be discovered during the course of routine examination (Barber, 1932; Sanguily and Blanco, 1945).

On clinical examination there are few signs of significance usually detected, except the presence of a palpable abdominal mass. Patients are in good general health, and if a benign tumour of the stomach is suspected, should certainly be examined for evidence of von Recklinghausen's disease. A secondary anæmia will probably exist owing to the recurrent hæmorrhage, and tests for occult blood will usually be positive. Performance of test-meals is of little help in the diagnosis. These patients will probably be submitted to gastroscopy and the presence of a benign tumour suspected. Paul and Chapman (1945) describe a case where the diagnosis was suspected at gastroscopy.

X-ray Examination. — Most endogastric tumours will be demonstrated by barium-meal examination. Moore (1924) states that no X-ray sign of benign tumour is pathognomic. A sharply defined filling defect, with a smooth surface, will be demonstrated in most cases, a large number showing a punched-out niche which is very characteristic of benign submucosal tumours of the stomach, though not of these tumours in particular. No evidence of infiltration of the gastric walls is seen and the mucosal folds appear normal on screening. The above signs are sufficient to warrant a diagnosis of a benign tumour of the stomach, but it is not possible to differentiate them by this means. Chaffin (1938), quoted by Schindler (1946), reported a case where the central ulceration in a benign tumour of the stomach, recognized on X-ray examination, was interpreted as being evidence of sarcomatous degeneration. This is an incorrect interpretation, the central *en face* niche being characteristic of all benign submucosal tumours. The presence of several irregular flecks of contrast material, indicating several ulcers present, might however suggest the presence of malignancy and a breaking down of the tumour.

Diagnosis and Differential Diagnosis.—In the absence of von Recklinghausen's disease, the final diagnosis of these cases will probably not be made until microscopical examination of the tumours has been performed, though their presence may be suspected. Benign tumours of the stomach should certainly be borne in mind when investigating cases of obscure hæmatemesis or melæna. A palpable abdominal mass will need differentiation from such conditions as carcinoma of the stomach, pancreatic cysts, retroperitoneal tumours, movable kidney, and enlarged spleen and gall-bladder. The X-ray examination may suggest the diagnosis of benign tumour and gastroscopy certainly so. If found incidentally at laparotomy, their nature should be appreciated and they should receive appropriate treatment.

Treatment.—If suspected from clinical examination, these cases should certainly be submitted to laparotomy, especially in view of the possibility of malignant change referred to. Wide surgical excision is advisable, though local excision may be preferable for small tumours. Local removal and enucleation is recommended by Schrager (1939). Geschickter (1935, a), and Cutler and Gross (1936, a), in discussing surgical treatment of peripheral nerve tumours, advise conservative resection for neurilemomata, more radical excision for malignant neurofibromata. Local excision is certainly to be avoided for both types in the stomach, as there is a considerable tendency to recurrence following incomplete removal. Partial gastrectomy will usually be necessary, the extent of the resection depending on the size and situation of the tumour. If the tumour is excessively large or proximally situated, even total gastrectomy is justifiable. This was performed in one of the cases reported by Ransom and Kay (1940). With the above treatment the prognosis is excellent and even in malignant cases is relatively good.

CASE REPORTS

Case 1—

HISTORY.—W. J. L., ages 62, a motor vehicle examiner, first attended hospital on July 1, 1946, complaining of pain in the epigastrium and occasional hæmatemeses. He had been treated by his private doctor with medicine, with some improvement. The pain when present was of a continuous grumbling character, with periods of freedom of as long as four weeks at a time. In May, 1946, he passed a black motion and had done so on several occasions since. His doctor referred him to hospital, where X-ray examination showed what was thought to

be a carcinoma of the stomach and he was referred to the Royal Cancer Hospital. He had lost a stone in weight, but had been gaining again recently. Appetite good. He had never vomited but had had occasional nausea. Bowels had been slightly constipated for some time. Micturition: some difficulty in starting.

Past History.—Appendicitis, treated conservatively, 1928.

Family History.—Nil relevant.

ON ADMISSION.—

General Condition: Very good. Well-covered man. No evidence of von Recklinghausen's disease.

FIG. 192.—*Case* 1. Radiograph of barium meal examination of stomach. The smooth filling defect is well shown on the lower part of the body of the stomach. The central niche is not well demonstrated, but was clearly seen on screening.

Tongue, Throat, and Fauces: Teeth all false. Nil abnormal.

Abdomen: Ill-defined mass palpable in right hypochondrium. Nil else abnormal. Rectal examination: Prostate uniformly enlarged.

INVESTIGATIONS.—

Blood: Hb. 93 per cent; Blood group B. Blood-urea, 35 mg. per 100 c.c.

X-ray: Barium meal (*Fig.* 192): "There is a large tumour on the lesser curvature of the lower part of the body of the stomach with an erosion in the centre of it. This is presumably a large malignant neoplasm, but in view of the smooth edges there is a chance that it is a benign tumour."

Fractional Test-meal: Free HCl and bile absent. No other significant findings.

OPERATION (July 11).—Laparotomy. A smooth tumour was found situated in the lower third of the stomach. One soft gland palpable overlying the head of the pancreas. Retrocolic antiperistaltic partial gastrectomy performed.

PROGRESS.—Convalescence uneventful. Patient discharged July 26. The patient has been seen on several occasions since. His condition is satisfactory and he is symptom-free.

FIG. 193.—*Case* 1. Showing smooth oval shape of tumour with central punched-out ulceration on mucous surface.

FIG. 194.—*Case* 1. Photomicrograph of representative tissue of tumour (× 30) showing typical elongated cells arranged in palisades.

PATHOLOGICAL REPORT (*Fig.* 193).—The surgical specimen presented a large intramural tumour mass, 9 cm. in diameter. This was situated submucosally and projected into the lumen of the viscus. Towards the pyloric extremity of the ovoid mass there was a crateriform ulcer. Several representative portions of the tumour mass were selected and prepared for microscopical examination.

Histology (*Fig.* 194).—The histology is that of a neurilemoma or schwannoma. It consists of interlacing whorls of fusiform cells, the nuclei of which in certain areas show well-defined palisading. The tumour is

intersected by relatively scanty and delicate fibrous and collagenous trabeculæ. The malignant potentialities of the tumour are indicated by numerous mitotic figures, nuclear hyperchromia, reduplication and syncitium formation, and by infiltration and destruction of the muscular coats of the stomach wall. The tumour may be classified as a malignant neurilemoma.

Commentary.—This case shows many of the typical features of neurilemoma of the stomach unassociated with von Recklinghausen's disease. The history of hæmatemesis and melæna, associated with little deterioration of the general health, led to X-ray examination of the stomach, when the presence of a benign tumour was definitely suspected from the smooth filling defect and the punched-out central ulceration, well shown on screening but not well demonstrated in the X-ray plate which is reproduced. The central ulceration is well seen in the excised specimen (*Fig.* 193). The patient was unfortunately not submitted to gastroscopy.

Histological examination of the tumour showed the typical characteristics of neurilemoma, part of which, showing the well-marked palisading, is reproduced (*Fig.* 194). Malignant changes had occurred in the tumour, and on pathological grounds it must be classified as a malignant neurilemoma or neurilemosarcoma.

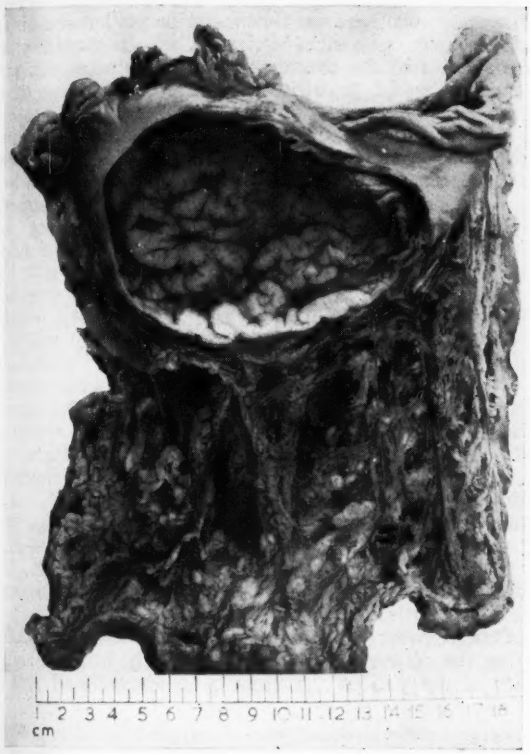


FIG. 195.—*Case* 2. Showing the interior of the specimen. The lobulated polypoid mass is well shown. The pedunculated part of the tumour prolapsing through the pylorus and causing obstructive symptoms and hæmorrhage is only partly visible.

FIG. 196.—*Case* 2. Posterior aspect of specimen, showing the subserous nodules, one of which has been cut for histological examination. Several enlarged glands are seen in the omentum.

Case 2.—

HISTORY.—V. S., aged 33, a married housewife, attended hospital on Sept. 26, 1936, complaining of abdominal discomfort, chiefly on the right side radiating to the right inguinal region. The pain had been present for three days, was unrelated to meals, and was relieved by taking alkalis. She had vomited on one occasion. Mild abdominal pain of a similar character had been present for six years intermittently. Bowels, normal; micturition, normal; menstrual history, normal. Aug. 8, 1936: Patient had retroverted uterus reduced under general anæsthesia, following miscarriages in March and June, 1936. It was then noticed that she had an unexplained anæmia. No hæmatemesis or melæna. Weight steady. Appetite fair. Recently the patient had felt extremely weak. She was thought to be probably suffering from chronic appendicular disease and her anæmia to be due to her recent miscarriages.

Past History.—Nil relevant.

Family History.—Nil relevant.

ON ADMISSION.—

General Condition: Good. Pale and listless. Temperature, 98·4°; pulse 88.

Mouth, Tongue, and Fauces: Tongue slight fur and dry. Teeth satisfactory. Nil else abnormal.

Abdomen: A few 'pigmented nævi' present. Tenderness on deep palpation to the right of the umbilicus and marked tenderness on deep palpation over McBurney's point. No mass palpable.

Rectal examination, nil abnormal. Vaginal examination, nil abnormal.

Heart and Lungs: Apical systolic murmur. B.P. 130/70. Some impairment of percussion note at right base, with fine râles on auscultation.

Central Nervous System: Nil abnormal.

INVESTIGATIONS.—

Barium Meal: As the patient was thought to be suffering from chronic appendicular disease the stomach was unfortunately not X-rayed but a pathological appendix was thought to be present on screening.

Blood: R.B.C. 2,520,000 per c.mm. Hb 36 per cent. Colour index 0·72. W.B.C. 10,800 per c.mm. Oct. 10, 1936: R.B.C. 3,000,000 per c.mm.; Hb 54 per cent.

W.R. negative.

Urine : Trace of albumin, few pus cells, culture sterile.

Fæces : Nil abnormal. Occult blood not performed.

The diagnosis was thought to be chronic appendicitis and it was decided to perform a laparotomy. The patient's anæmia was attributed to her recent miscarriages.

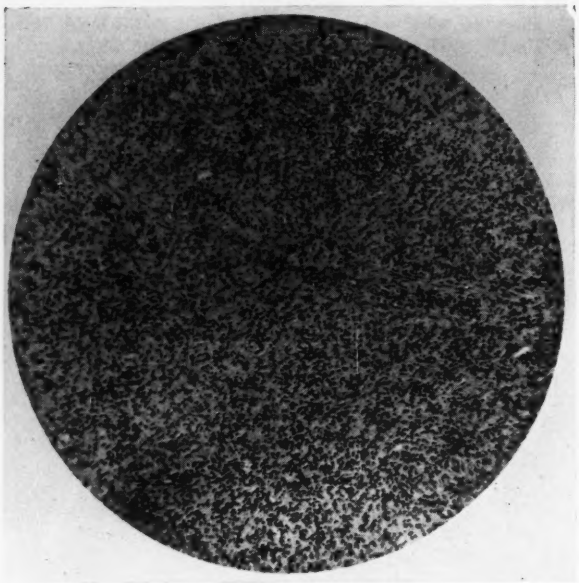


FIG. 197.—Photomicrograph of representative part of tumour from *Case* 2 (× 30) showing absence of typical palisading. Interlacing strands and whorls of elongated spindle cells with much fibrous tissue present, but no obvious specific distinguishing features.

OPERATION (Oct. 21).— Laparotomy. Abdomen opened through right paramedian incision. Appendix found to be normal and removed. On examination the stomach was found to contain a soft fleshy mass which was prolapsing through the pylorus. This was thought to be probably a sarcoma of the stomach. Partial gastrectomy was performed, three-fifths of the stomach being removed.

Convalescence uneventful. Patient discharged Nov. 10, free from pain.

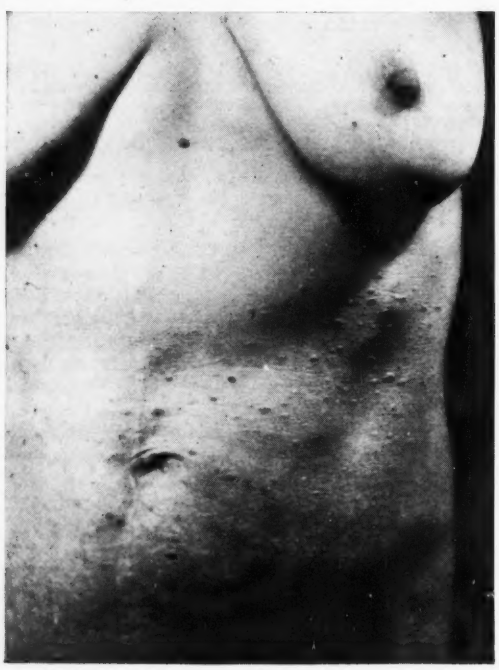
PATHOLOGICAL REPORT (*Fig.* 195).—Grossly, the interior of the stomach presents the appearance of hypertrophic polypoid gastritis, the stomach containing a lobulated polypoid mass, one large pedunculated part of which is projecting freely into the pyloric antrum and is ulcerated. Several subserous nodules were found on the anterior wall and on the posterior wall three similar nodules were present (*Fig.* 196). Soft elastic glands were present in the omentum. The diagnosis of hypertrophic polypoid gastritis is untenable in view of the subserous nodules.

Histology (*Fig.* 197).—The gastric mucosa showed chronic hypertrophic gastritis. The subepithelial zones of the muscular coats were diffusely infiltrated by a highly cellular tumour. This presented the characteristics of a neurofibroma. Nuclear palisading was distinctly discernible, but was less marked than in the neurilemoma. It is probable that this lesion is a local manifestation of von Recklinghausen's neurofibromatosis.

PROGRESS.—The patient was traced for the purpose of writing this paper, and was seen on April 28, 1947, approximately eleven years since her first attendance.

She was a healthy looking woman of normal physical development. Mentally alert. No skeletal changes present. She had had no gastro-intestinal symptoms since her operation. Her lower chest, shoulders, and back were found to be studded with typical subcutaneous nodules of von Recklinghausen's disease (*Figs.* 198, 199), which she stated started to appear soon after her operation in 1936. No nodules were found on the limbs, head, or neck, and no areas of pigmentation were present. The abdominal wound was well healed and nothing abnormal was found on examination of the abdomen.

Commentary.—This tumour was found incidentally during the course of laparotomy, though, had the significance of the patient's anæmia been appreciated and the



FIGS. 198, 199.—*Case* 2, eleven years after the removal of the gastric tumour, showing the typical nodules of von Recklinghansen's disease on the trunk, abdomen, and back.

stomach been X-rayed or gastroscopy performed, the intragastric tumour would no doubt have been discovered. The anæmia was attributed to the patient's recent miscarriages, though it was undoubtedly due to chronic gastro-intestinal hæmorrhage from the pedunculated part of the tumour mass prolapsing through the pylorus.

The pathological diagnosis of the tumour was certainly in doubt and no evidence of von Recklinghausen's disease was present when the patient was first seen, though the presence of subserous nodules in the stomach wall were certainly suggestive of the diagnosis.

It can be seen from the photomicrograph (*Fig.* 197) that the histological characteristics of the intragastric neurofibroma are far less well defined than those of the neurilemoma. Palisading is absent and the neurogenic origin of the tumour is less obvious. The subsequent clinical findings on follow-up of the patient finally proved the tumour to be a neurofibroma of the stomach associated with von Recklinghausen's disease beyond all possible doubt. There was no evidence of sarcomatous change in the tumour.

SUMMARY

1. A short account is given of the neurogenic tumours occurring in the stomach; their histogenesis, classification, pathology, and incidence are discussed.

2. Two types of benign nerve-sheath tumours of the stomach are recognized: (*a*) Neurilemoma; (*b*) Neurofibroma associated with von Recklinghausen's disease.

3. The clinical features and diagnosis of the tumours are described.

4. Two cases of tumours of neurogenic origin occurring in the stomach are reported.

My thanks are due to Mr. R. C. B. Ledlie for his kind permission to publish these two cases and to Dr. Woodhouse Price for the pathological reports.

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THE BILHARZIAL URETER

SOME OBSERVATIONS ON THE SURGICAL PATHOLOGY AND SURGICAL TREATMENT

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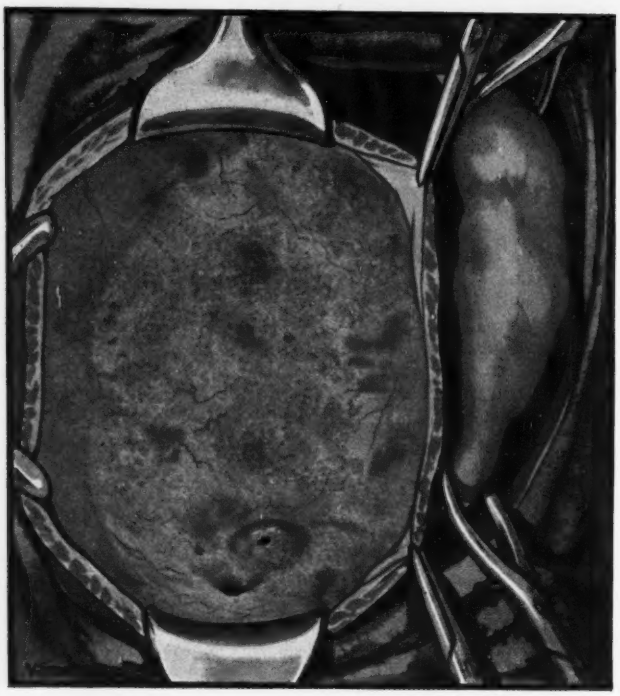
SURGICAL PATHOLOGY

Mode of Infection of the Ureters.—A study of the anatomy of the mesenteric veins and some observations on the results of their injection in the dead body by Dr. Boulgakouf, Dr. B. Salib, and myself, seem to shed some light on the usual channels traversed by the bilharzia worms in their passage from the portal system to the ureters.

After injection of indigo-carmine into the superior mesenteric vein the existence of collateral venous branches passing from its radicles in the cæcum to the right ureter could be made out clearly. Similar injections into the inferior mesenteric vein also showed free anastomosis between its branches and the veins of the left ureter.



On both sides these anastomoses occupied an area corresponding to that part of the posterior wall of the abdomen which lies lateral to the third and fourth lumbar vertebræ in the fascia transversalis. The anastomoses on the left side of the body were more extensive and marked than those on the right.

Apart from this set of mesenterico-ureteral anastomoses, the portal and systemic veins communicate in a plexus of veins which overlies the last two to three inches of the pelvic portions of the ureter, the middle rectal veins anastomosing with veins coming from the above-mentioned segment of the ureters and also with veins issuing from the vesico-prostatic venous plexus.

The bilharzia worms, mostly *Schistosomum hæmatobium*, travel against the blood-stream and reach by their own efforts the walls of the ureters, the submucous layers in particular. Their ova pass from here mainly in the direction of the ureteric lumen and, probably as the result of the irritation of a miracidial secretion, some of the histiocytes of the body, e.g., monocytes, eosinophils, fibroblasts, and sometimes giant cells, also collect around one or more bilharzia ova to form a bilharzial unit—a " bilharzioma " (Sorour) or " bilharzon " (Makar).

The different bilharzial lesions formed from these bilharzial units belong to one of two main pathological types :—

1. *Hyperplastic* : in which the presence of " bilharzons " in the epithelium of the ureter and underlying connective tissue produces nodules and papilloma-like bodies. Here the epithelial hyperplasia seems to follow the path of least resistance, i.e., away from the basement membrane towards the lumen. Papillomata of the ureter are smaller in size and not so friable as those found in the bilharzial bladder, although when the tube is markedly dilated they may attain fairly large dimensions and acquire softer consistency (*Fig.* 200). At first vascular and soft, they later become tough, and frequently undergo calcification which may be so marked as to convert them into stone-like bodies (*Fig.* 201).

FIG. 200.—Bilharzial ' sandy bladder ' with contracted distorted trigone and bilharzial left ureter, with a large papilloma (*shown also above*, × ⅔).

Sometimes the epithelial hyperplasia extends its activity in the opposite direction, out towards the submucosa among the cells of which tubular or ' flask-like ' epithelial growths proceed to form, as Ferguson and Urquhart have shown, pseudo-glandular structures. Occlusion at their necks transforms them into a kind of retention cysts containing cellular debris, colloid material, and blood-pigment. The position of these cysts varies as regards their relationship to the ureteric mucosa, sometimes appearing as vesicles perched on its surface (*Fig.* 202) ; at other times they lie underneath the mucous membrane which thereby bulges towards

the lumen, while in other cases small dark-brown pits, peppered irregularly over the surface, are the only indication of their presence (*Fig.* 203).

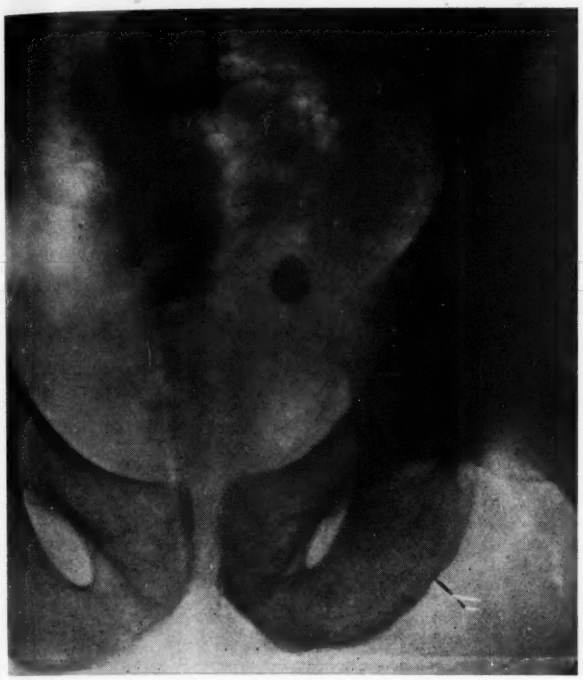


FIG. 201.—Showing calcified bilharzial papilloma in the left ureter simulating ureteric calculus.

The cysts vary in size from a tiny speck to that of a pea, and are found particularly in those dilated parts of the ureter that lie proximal to strictures, especially about the levels of the ischial spines and that of the third lumbar vertebra. This cystic condition, so-called " bilharzial ureteritis cystica ", is more frequently seen in the ureter than in the bladder.

Epithelial hyperplasia in the ureter very rarely passes on to malignant carcinomatous changes, and so far only two examples of bilharzial carcinoma of the ureter have been met with by the writer (*Figs.* 204–206).

2. *Hypoplastic :* Sooner or later the fibrosis formed around and within the bilharzial infiltrations kills their incarcerated miracidia. With the cessation of the irritation caused by their secretion, the process of hyperplasia in the epithelium usually comes to a standstill ; that of fibrosis proceeds further, and calcification in the bilharzial ova ensues. The mucous membrane covering such ova undergoes marked atrophic changes until only a thin layer of fibrous membrane covers the underlying calcified bilharzial ova. This condition is often seen on opening the ureter at operation and gives its mucous membrane a wash-leather-like appearance and imparts a bloodless lifeless touch under the cutting knife. To this appearance the well-known term ' sandy patches ' has been applied (*Fig.* 207).

The atrophy of the epithelium may go so far as to lead to its complete desquamation over areas of varying sizes.

It is common to find such denuded surfaces in the ureter carpeted with a granulomatous tissue formed of capillaries sprouting from the subepithelial

FIG. 202.—Bilharzial ureteritis cystica. Bilharzial stricture of intramural portion of the ureter and dilatation in its pelvic portion which harbours the cysts. (*From a museum specimen.*)

vessels. These granulomata occur in different stages of maturity and present nodules and masses of varying shades of colours from the fresh rosy

FIG. 203.—Flask-like epithelial invaginations into the submucosa. (*After Ferguson.*)

colour of young capillaries to the old grey of scar tissue.

Deposition of reddish crystals of urates, black concretions of oxalates, or whitish encrustations of phosphates on the granulomatous surfaces may take place. From these deposits concretions may be formed and are frequently seen in the dilated parts of the ureter. They are of a composite chemical nature with phosphates predominating; their nuclei may contain bilharzial ova (*Fig.* 208).

Bilharzial ulcers are not so common in the ureter as they are in the bilharzial bladder. This relative immunity is probably due to the fact that the latter organ is more exposed to trauma and infection than the ureters. Nevertheless, superficial small ulcers may be encountered in the ureters, and are probably due, as suggested by Aly Ibrahim, to the spontaneous sloughing of some ureteric papillomata.

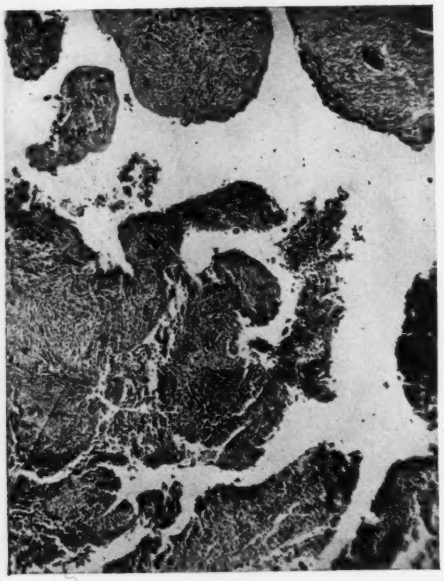


FIG. 204.—Bilharzial epidermoid carcinoma of lower end of left ureter. Note calcified ova (*B. hæmatobium*).

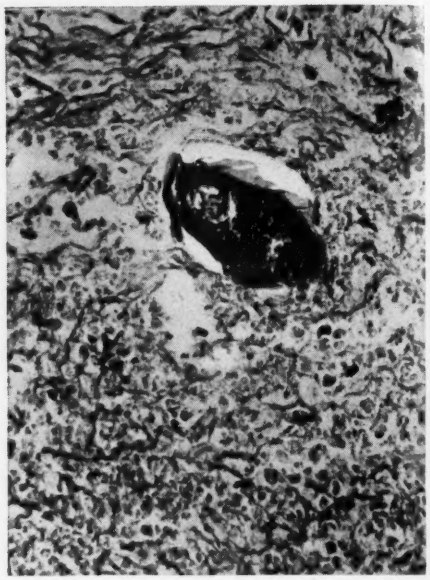


FIG. 205.—Bilharzial ova with cancer cells around it. (Magnified view.)

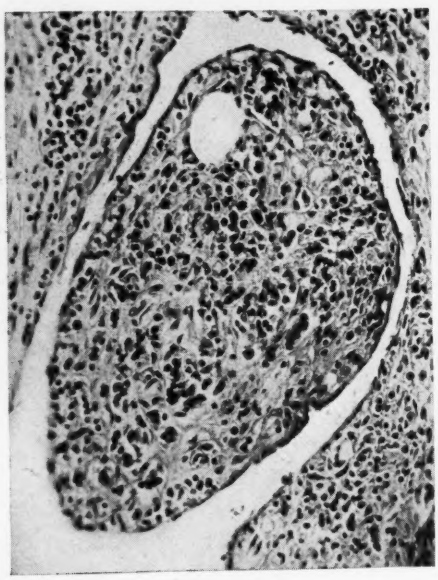


FIG. 206.—Cancerous embolus in a vein of lower end of left ureter.

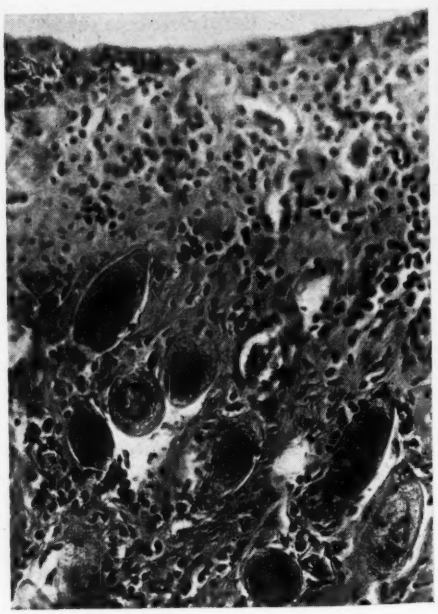


FIG. 207.—Microphotograph of a bilharzial 'sandy patch' showing atrophied mucosa, bilharzial ova, eosinophils, and fibroblasts.

Secondary infection is a frequent complication; this helps to aggravate the ulcerative processes and may spread to the deep parts of the ureteric wall or may even reach the periureteric tissues. Rarely, it may give rise to periureteric suppuration, and such abscesses have been known to open into the neighbouring organs, e.g., the rectum or vagina;

recto-ureteric and uretero-vaginal fistulæ may thus be formed, but the latter are not so common as similar bilharzial fistulæ connected with the bladder.

However, the common result of the bilharzial infiltration of the ureter is a dense fibrosis involving all or most of its layers. Its contraction leads to stenosis of the tube and even to complete obliteration of its lumen.

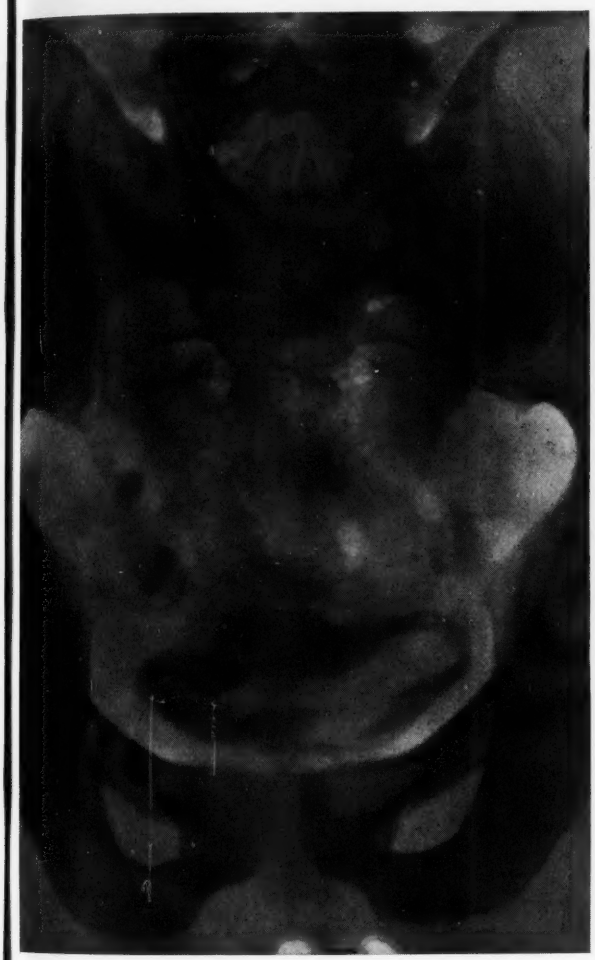


FIG. 208.—Showing concretions formed within the pelvic portion of the right ureter. Marked bilharzial calcification is seen in the bladder wall.

The above pathological lesions may occur in part or the whole of the ureter. The disease may be limited to one ureter, but quite commonly it is bilateral. Usually such lesions are seen in one or the other of the following segments of the ureter :—

a. The intravesical portion of the ureter, where the ureteric affection is but a part of the vesical bilharziasis. Cystoscopy reveals its presence in different forms : thus, the ureteric orifices may be narrowed, deformed, or displaced ; the lip of the orifice may be covered with bilharzial nodules or papillomata which may reach down into the bladder, hanging from a prolapsed ureteric mucosa. The rigid terminal parts of the ureters may look like pipes through which urine flows passively into the bladder, the regular rhythmic efflux being absent. The passage of a ureteric catheter in such cases, even when of a small size, is often very difficult. Its tip may be pushed up for about a centimeter proximal to the ureteric orifice and its passage is then arrested.

Due to atrophy of their muscles and rigidity of their walls the valvular action of the intramural portions of the ureters is sometimes completely lost, and fluids injected into the bladder may find their way to the kidneys (*Fig.* 209).

b. The second common site of bilharzial infiltration of the ureter is that length of the canal which

FIG. 209.—Showing regurgitation of opaque fluid, injected in the bladder, back through the ureters.

lies between the bladder wall and the level of the ischial spine. Stenosis of this part of the tube is not so frequent as that of the intramural portion. One more often finds this segment dilated from the back pressure caused by stenosis of the intravesical part : indeed, dilatation of this portion of the ureter alone is so constant as to merit a special diagnostic value. Such localized ureteric dilatations have been considered by Clement and other urologists of South Africa as pathognomonic of bilharziasis of the ureter. Our experience in this country strongly supports this remarkable observation (*Fig.* 210).

c. That part of the ureter which corresponds to the third lumbar vertebra is less frequently the seat of bilharzial lesions, particularly papillomata and stenosis.

The existence of well-developed mesenterico-ureteric anastomoses at this level may account for this localization of bilharzial lesions. Again the fact that such venous anastomoses are more free on the

left than on the right side may explain why these bilharzial lesions are commoner on the left side of the body (*Figs.* 211, 212).

d. Lastly, the whole ureter may be involved in the disease on one or both sides of the body. Recurrent, perhaps also intensive, bilharzial infections may account for the severity of such lesions (*Fig.* 212).

The ultimate result of the bilharzial ureteric involvement, wherever the site may be, is a gradual dilatation of the tube proximal to the lesion, and

FIG. 210.—Showing a dilated bilharzial right ureter and a normal left both filled with uroselectan.

extending from below upwards. The ureteric muscle undergoes at first compensatory hypertrophy which slowly passes into the reverse condition of atrophic dilatation until the whole tube is enlarged, lengthened, and made tortuous. These changes may be of such degree as to make the ureter resemble the intestinal tube. Calcification may also involve a part or the whole of its length (*Fig.* 212).

Chronic plastic periureteritis leading to fibrosis and thickening of the neighbouring periureteric tissues occurs in a marked degree in the pelvic segments and to a lesser extent more proximally. The distal portions of the tubes become so deeply buried in the dense fibrous tissue that their identification or manipulation often presents great difficulty to the surgeon.

Distension of the pelvis of the kidney and later on of its calices also—i.e., pelvic and renal hydronephrosis—follow, which do not differ in their pathology from that of the non-bilharzial conditions, though the changes take place more gradually. Supervention of secondary infection expedites their

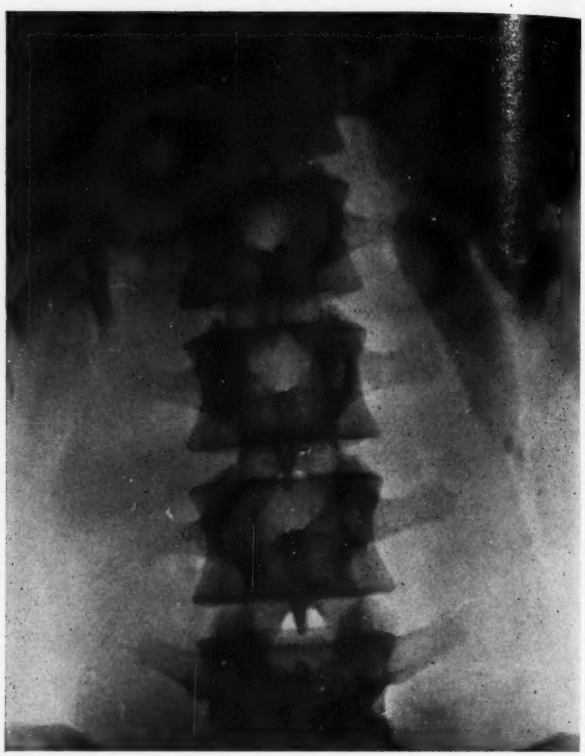


FIG. 211.—Showing bilharzial stricture of the left ureter opposite the third lumbar vertebra. A small stone lies just above the stricture.

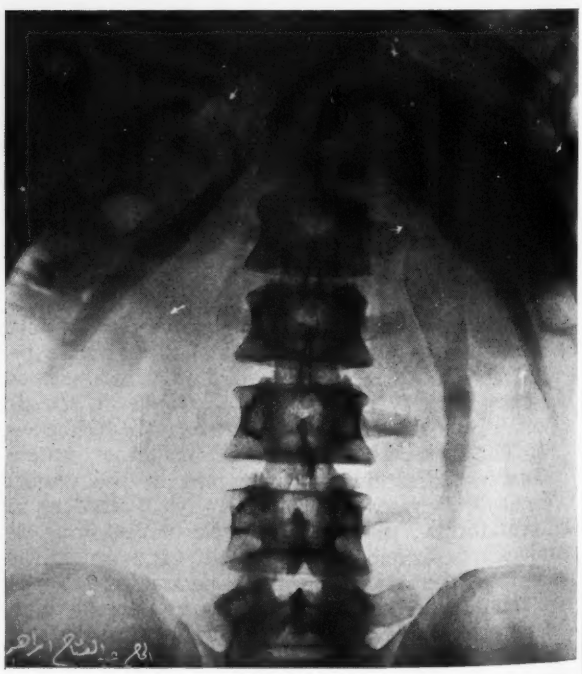


FIG. 212.—Plain radiograph showing bilharzial calcified left ureter and left renal pelvis. Note severity of obstruction opposite third lumbar vertebra.

evolution. Bilharzial hydro-ureter and hydronephrosis are usually bilateral, the difference being only one of degree; but sometimes advanced hydro- or pyo-nephrosis may be strictly limited to one side only, a well-functioning or even normal kidney being present on the opposite side.

SURGICAL TREATMENT

Specific antibilharzial treatment and that of any superimposed secondary infection should precede the surgical treatment, the latter varying according to the characteristics of the lesion in question. Thus:—

a. Strictures limited to the terminal inch of the ureter may be dilated by stiff ureteric bougies introduced through a catheterizing cystoscope. This method has a limited scope, since most of the bilharzial strictures met with in Egypt are so dense that their dilatation requires force and an amplitude of manœuvring that neither the bilharzial bladder nor the cystoscope can sustain with safety. Again, in view of the weak muscular contracting force which bilharzial ureters possess, and especially their dilated segments that lie proximal to such stenoses, obviously dilatation of these strictures must be carried out fully before they can be expected properly to drain the urine from the corresponding renal pelvis and ureter.

Dilatation of the stenosed portion of the ureter which extends above the bladder is carried out after a ureterostomy performed just proximal to the stricture, which is then dilated by curved Hegar's dilators in a manner similar to that used for dilating the cervix uteri; in very tough strictures a fine tenotomy knife may serve as a ureterotome. By cutting through the dense scar tissue in one or more planes, dilatation is made possible. A fine rubber catheter is then passed for a few inches proximally, its other end being pushed into the bladder. This is kept in place for about a week, at the end of which the catheter is removed through the cystoscope.

In cases where the ureteral stenosis is complicated by other intravesical bilharzial lesions, the bladder is opened after securing the stenosed ureter above it. Intra-ureteric and intravesical manipulations can thus be combined with advantage for restoring the patency of the ureter and dealing with the vesical lesion.

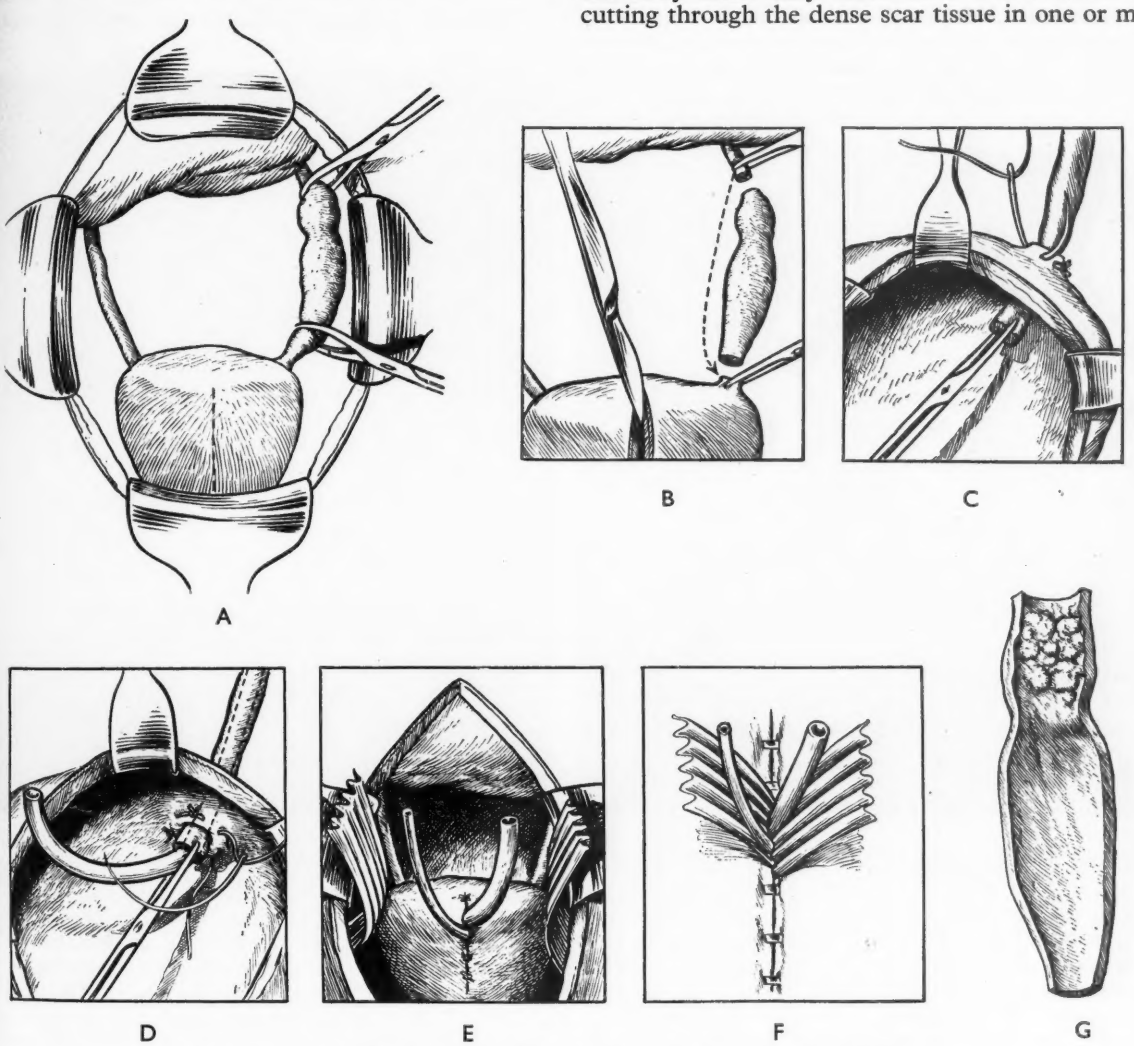


FIG. 213.—A–F, showing the steps in the operation; G, The growth in the ureter.

Excision of the stricture, followed by end-to-end anastomosis, is an excellent procedure, but is only applicable to more proximally situated lesions, e.g., those found in the upper pelvic and lumbar region.

b. Implantation of the ureter into the bladder, after resection of its stenosed segment, is indicated in severe cases in which none of the above surgical procedures can be practised.

The tube is usually so lengthened and relaxed by disease that with a little dissection it can be brought down in the majority of cases to the level of the trigone after excising its obliterated two to three inches. The method followed by us in performing the ureteric implantation is briefly as follows: Under spinal anæsthesia the ureter is reached extraperitoneally through a suprapubic midline incision. The hard fibrosed tube is followed into the substance of the bladder and is dissected free of the latter until the vesical mucosa is reached. The bladder is opened in the middle line and the terminal attachment of the ureter cut into by vision from without inwards. A curved forceps is next passed from the bladder into the tunnel created by the removal of this segment of the ureter. The stenosed part of this is excised, a fine rubber catheter is passed proximally into the ureter for a few inches and is fixed to the ureteric wall by one fine non-chromicized catgut stitch. The other end of the catheter is caught with the forceps and passed through the tunnel into the bladder and out through the cystotomy wound. By pulling on the catheter the ureter is drawn into the bladder until about ½ in. of its tip lies freely intravesically and is then anchored to the mucosa and submucosa by chromicized fine catgut at four points. A similar anchorage is effected between the superficial bladder muscular fibres and the wall of the ureter on the outside of the bladder without penetrating the ureteric mucous membrane (*Fig.* 213). The bladder is closed around a drainage-tube and also around the intra-ureteric catheter, and the supra- and infra-vesical spaces are drained by corrugated rubber for two to three days. The catheter and tube are removed from the ureter and the bladder after a week.

The dissection of the terminal portion of the ureter completely out of the bladder is a tedious process, but it gives the implanted ureter a position and course as near to the normal as possible (*Figs.* 214, 215). It also ensures complete removal of the diseased obstructed ureteric tissues. The importance of these points becomes clear if it be remembered that such bilharzial bladders are often reduced in their capacities and are mostly soiled with secondary infection.

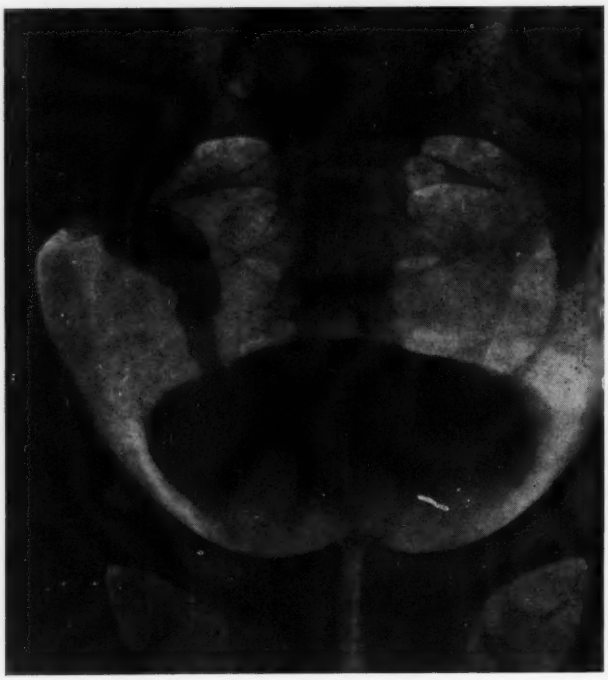


FIG. 214.—Radiograph showing bilharzial stenosis of the left ureter (before operation).

FIG. 215.—Radiograph showing condition after operation (implantation of the left ureter into the bladder).

The method is preferable to that in which, after ligating the cut lower end of the stenosed segment without resecting the latter out of the bladder, the new ureter is implanted above the level of the trigone, directly, without giving it its fully oblique normal course.

c. Bilharzial Papillomata.—Those lying at the ureteric orifices, or arising a little more proximally and peeping down through them into the bladder, can be fulgurated by a cystoscopic diathermy electrode. When they lie at higher levels their excision is carried out after ureterostomy; the

longitudinal incision in the ureter is stitched up transversely or obliquely to guard against stenosis. Should the papillomata be numerous or of large size the portion of the ureter harbouring them would have to be resected and the two ends of the tube anastomosed (*Fig.* 216). This method is applicable

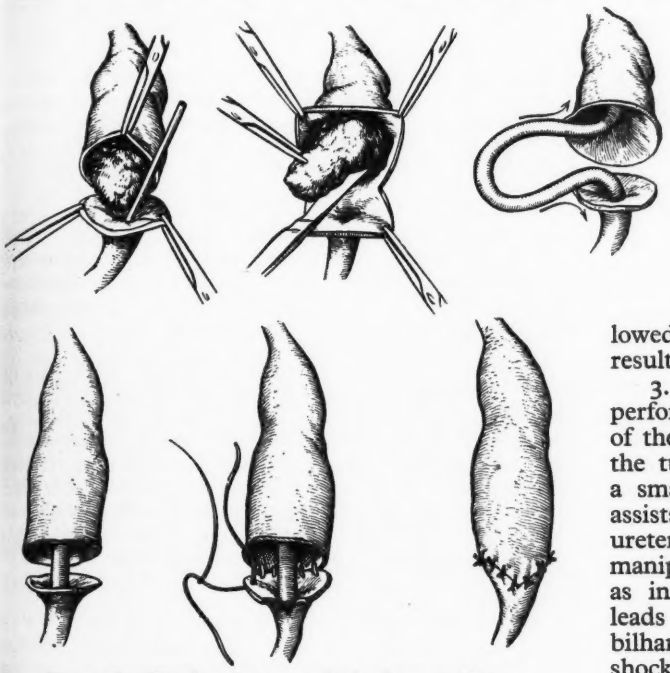


FIG. 216.—Showing the removal of a large papilloma by resection and anastomosis.

to cases of highly situated tumours, e.g., in the lumbar or upper pelvic segments of the ureter. If they be situated very low down the pelvis implantation of the ureter in the bladder after excision of the papilloma-bearing area would have to be done (*Fig.* 213).

Nephrectomy.—Surgical teaching formerly frowned upon nephrectomy in bilharzial ureteritis. While this view is still true of the great majority of cases (about 90 per cent), on the other hand where the bilharzial lesion is strictly unilateral or the other kidney is only very mildly affected with bilharziasis, nephrectomy seems justifiable provided that none of the various surgical procedures mentioned above can be practised with any hope of success.

RESULTS

The total number of cases on which this article is based is 30, a number too small to warrant giving any conclusions or even to help in assessing the values of the various surgical procedures adopted.

However, as this is a preliminary note on the subject the following impressions may be given :—

1. When the results of operative and non-operative methods of treatment are compared, the balance is definitely in favour of operative intervention, especially in severe bilharzial lesions.

2. Of the different surgical procedures, excision of the bilharzia-affected parts followed by ureteric anastomosis has given the best results and the least post-operative trouble.

3. Implantation is well worthy of trial and its performance should be encouraged. The difficulty of the operation is mainly due to the embedding of the tube in fibrous periureteritis. The passage of a small-calibred ureteric catheter before operating assists identification and the digging out of the ureter from the depths of the pelvis. Gentle manipulation and patience are as necessary here as in other difficult dissections. Rough handling leads to severe hæmorrhage and laceration of such bilharzial non-elastic structures. Post-operative shock, infection, leakage of urine, uræmia, or secondary hæmorrhage—one or more of these are only too likely to follow athletic surgery.

In well-chosen cases the results justify the operation with all its difficulties.

4. Dilatation of the stricture should be carried out diligently until the calibre of the stenosed segment is made sufficiently wide in order to counteract the remarkable tendency to reclosure of dilated strictures. This demands patient work without force, and even when these and other ideal conditions are fulfilled, recurrence of the stenosis cannot be obviated with certainty.

Rapid dilatation of the last ureteric inch by means of Buerger's or other dilators, introduced through the cystoscope, has given us only temporary satisfaction.

VENOUS OBSTRUCTION IN THE UPPER EXTREMITY

By E. S. R. HUGHES

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PAGET (1875) and Schrotter (1884) both described a condition wherein an arm of an otherwise perfectly healthy individual became diffusely swollen and discoloured. Schrotter believed that movements of the shoulder girdle damaged the axillary vein and were thus responsible for the formation of a thrombus.

This theory has been accepted by many and hence the term 'traumatic thrombosis of the axillary vein'. But as neither trauma nor thrombosis is a constant feature and since the site of obstruction is still uncertain, a more general designation has been preferred, such as 'intermittent venous claudication' (Lohr, 1933; Harms, 1938) and 'venous stasis' (Hauge, 1942).

The association of acute venous stasis and a healthy person constitutes a syndrome which, in the absence of unanimity of opinion as regards the aetiology or pathology, might well be called the 'Paget-Schrotter syndrome'.

CASE REPORTS

Case 1.—Miss F. C. In 1923, at the age of 22 years, she was employed cleaning floors. One day she stopped to have a cup of tea. She put her right hand on the table and discovered that it was blue and swollen from the knuckles to the shoulder. In the evening she consulted her doctor, who advised her to rest it in a sling. There was no improvement and a fortnight later she was put to bed at hospital, where she remained for three weeks. The swelling and blueness subsided a little, but not completely. About two months after the onset of the condition, she began to experience a deep dull aching pain mainly above, but also below, the elbow.

Despite the lapse of twenty-five years she still complained of a dull aching pain in the upper arm; it quickly became blue in cool weather or on carrying the arm in a dependent position.

Examination revealed a healthy woman. Both arms were carried normally. There was slight limitation of the combined scapulo-humeral movements of the right shoulder, but otherwise the active and passive movements were normal. There was 1 cm. circumferential swelling of the right upper arm and 2·5 cm. of the forearm. The whole arm was more deeply coloured than the normal left arm. There was a marked varicosity of the superficial veins of the arm and the veins about the shoulder girdle were more apparent on the right side. The right hand was quite cold despite a high room temperature. There was tenderness along the course of the axillary and subclavian veins. A radiological examination and full blood analysis showed no abnormality and the Wassermann reaction was negative.

Case 2.—Mrs. A. S., aged 60 years. On Feb. 28, 1947, she finished her morning's housework and proceeded to wash before eating her lunch. She washed her arms and noticed no abnormality. She then put her left arm up and washed the back of her neck. After bringing it to the side again, she noticed that the upper part of the upper arm was quite blue. It remained that colour for the rest of the day and on waking next morning, she found that the whole of the left upper arm was reddish blue and swollen. By the afternoon the swelling had spread to involve the left forearm and hand and she experienced difficulty in holding a fork with the left fingers during the evening meal. She consulted her doctor, who ordered a sling and rest in bed. On March 4 she began to get a deep dull aching pain in the left arm near the shoulder, as well as in the forearm muscles.

After six weeks' rest in bed the pain, swelling, and discoloration lessened, and she was gradually able to resume her daily work. Twelve months later she still complained of a dragging pain in the axillary region and her fingers felt stiff.

Examination revealed a healthy, elderly woman. The left arm was slightly discoloured below the elbow. All the active and passive movements of the limb were normal. There was 1 cm. circumferential swelling of the left upper arm and 2 cm. of the forearm. There was some dilatation of the superficial collateral vessels coursing across the clavicle. The hand felt cold and dry. There was tenderness over the course of the axillary vein. The Wassermann reaction was negative and a radiological examination and a full blood analysis were normal.

Case 3.—Mr. T. M., aged 36 years. On May 27, 1946, the patient was carrying 2–3 cwt. of meat on his right shoulder, when he suddenly noticed that his right arm was swelling. There was no pain. He stopped work, but the swelling increased; the hand became a mauve colour and felt numb. He went home and on awaking next morning the patient found that the arm was worse and he went to hospital. He was treated with heparin; a total of 75,000 units was administered over a period of seven days. The patient discharged himself after two weeks in bed. He had improved very considerably, though the arm remained slightly swollen and the colour still somewhat blue. He did not resume his previous occupation, but after light odd jobs began chicken farming.

Eighteen months later he had no symptoms at all except when he unduly exercised the arm, which caused the limb to swell.

Examination revealed a healthy man of wiry build. The arms were held normally. The active and passive movements were full. The right arm showed a 2·5 cm. circumferential swelling of the arm above the elbow and a 2·5 cm. below the elbow; the hand was puffy over the dorsum. There were slight varicosities of the superficial veins of the right arm and dilatation of the superficial veins in the region of the shoulder girdle. The right hand and forearm were a deeper colour than the left and were definitely cooler. A radiological examination and full blood analysis revealed no abnormality; the Wassermann reaction was negative.

Case 4.—Mrs. E. H., aged 37 years. This patient was engaged in the ordinary duties of a housewife and enjoyed perfect health. On June 22, 1947, on awakening, her husband brought her some tea; he drew her attention to her left arm, which was swollen and blue. After dressing, her left arm began to ache; she consulted her doctor, who sent her to hospital the same day. She was given a sling and treated in the Out-patient Department.

Nine months later she still suffered pain in the left arm, particularly after any effort. It fatigued quickly and she could not carry any shopping on that arm.

Examination revealed a healthy individual. The arms were carried normally. The active and passive movements of both limbs were normal. There was a 2 cm. circumferential swelling of the left upper arm and 1 cm. in the forearm. The dorsum of the left hand was slightly swollen as compared with the right. The superficial veins in the region of the shoulder girdle were dilated and the external jugular vein was rather more prominent on the left side. There was a slight reddish-blue discoloration of the hand and forearm. The whole arm felt cooler than the unaffected arm. A radiological examination and full blood analysis revealed no abnormality and the Wassermann reaction was negative.

Case 5.—Mr. L. J. In February, 1941, when aged 38 years, the patient developed a dry cough, which was particularly severe at night time. Soon after he was trapped under a bombed building, and he was forced to push and strain to release himself. Two weeks after this experience and whilst rubbing his hands to keep himself warm he noticed that his right arm was larger than his left. In a few hours the swelling had increased to such a degree that it was difficult for him to remove his arm from his coat. His doctor immediately sent him to hospital, where he received in-patient treatment for seven weeks and was subsequently treated as an out-patient in the physiotherapy department for several years.

Seven years later the patient was suffering from much pain. He was unable to lie on his right side in any comfort. He found it difficult to shave and even cleaning his shoes with the right hand was a formidable task.

Examination revealed a healthy man. The arms were carried normally. Active and passive movements of both arms were normal. There was a circumferential swelling of 2 cm. in the arm above the elbow and 1·5 cm. of the forearm. The superficial veins of the arm were grossly dilated and collateral vessels, greatly distended, coursed over the shoulder girdle and over the chest wall. The whole arm was of slightly deeper colour than normal, but no temperature difference could be detected. There was no thrombosed vein palpable and the axilla felt

normal. A radiological examination and full blood analysis revealed no abnormality and the Wassermann reaction was negative.

CLINICAL FEATURES

The patients are typically active individuals, between the ages of 18 and 40 years. Of reported cases 80 per cent have been males and in 70 per cent the right arm was involved (Hughes, 1948). In many the onset has occurred during or soon after the performance of some strenuous work, but in others the alleged effort has been minimal or even absent, and in 10 per cent of cases the abnormality was first noticed on waking in the morning. The condition develops over the course of a few hours; in some cases the onset has been exceedingly rapid, whilst in others it has been rather more gradual.

Swelling is usually the initial symptom. It involves the whole of the arm from fingers to shoulder girdle and not infrequently the lower part of the neck is involved. The swollen arm is firm and rarely exhibits 'pitting' on pressure.

The skin assumes a slightly cyanotic hue, though on occasions the colour has been described as pink (Kaplan, 1938; Hoffert, 1942; Roelsen, 1944). The discoloration is usually diffuse but may be mottled, and is more obvious in the hand and lower part of the forearm.

The superficial veins are invariably more prominent than usual. The large veins, which are normally visible in the cubital fossa, become tense, compressible cords; whilst veins about the shoulder girdle become apparent for the first time as the collateral venous circulation develops.

Pain may be referred to the arm or to the shoulder-joint, particularly the axillary region. It is usually described as a dull aching pain and is of varying severity. There may be severer exacerbations radiating down the arm from the shoulder. There is often no pain at all, or it may only be experienced on exercise.

A firm tender cord is sometimes palpable in the axilla, at times extending down the inner side of the arm to the forearm. This occupies the position of the venous trunks and is usually regarded as a thrombosed vein, although this has occasionally been disproved by operative exploration. In others, apart from tenderness in the axilla, nothing abnormal has been found. Despite its more concealed position, similar clinical findings have been noted in the subclavian vein.

Direct radiological examination is almost invariably negative. It is an important investigation, however, as it helps to exclude cardiac disease and aortic aneurysm. On rare occasions a cervical rib has been discovered (Pinelli, 1932; McGoogan and Simmons, 1933; Andersson, 1938; Hermodsson, 1943).

Some authors have performed certain special investigations, but in so far as they prove the obvious, that is, the presence of venous stasis, their value is small. The measurements of venous pressure, oxygen saturation of venous blood, and circulation time have, without exception, confirmed the presence of this stasis. Venography enjoys considerable popularity, but it appears to have no more value than the above tests; collateral vessels, which clinically can always be seen, have been visualized in every case but one (Roelsen, 1939); the main venous trunks exhibit varying appearances which are usually interpreted as indicating obstruction. Arterial pressure and oscillometric recordings are of more academic than practical interest.

Almost always there is no systemic reaction whatever. With a few minor exceptions (Wilson, 1922; Gould and Patey, 1928; McGoogan and Simmons, 1933; and others) the temperature, pulse, white-blood cell-count, and sedimentation rate are normal. Apart from the case of polycythæmia reported by Stover and Herrel (1940), no cytological nor chemical defect of the blood has been found. In only the occasional case has evidence of specific infection been present (Delbet, 1913; Routier, 1913; Gautier, 1913; and others).

DIFFERENTIAL DIAGNOSIS

Signs of obstruction of the main venous outflow from the upper limb, in a perfectly healthy patient, leave little doubt as to the diagnosis. As the cardinal signs of venous stasis are exhibited by nearly every case, the problem resolves itself into distinguishing this condition from the sequelæ of such grave conditions as aneurysm of the aorta, metastatic carcinoma, and congestive cardiac failure.

An aortic aneurysm sometimes compresses the innominate vein, with congestion of both the subclavian and jugular systems. A positive Wassermann reaction will excite suspicion, and a radiographic examination of the chest will confirm.

Thrombosis of the axillary vein may complicate a carcinoma of the breast, or less commonly a primary carcinoma elsewhere when secondaries are present in the axilla, though this is often observed at some time after surgical removal when its appearance usually signifies recurrence or compression of the vein by scar tissue. The diagnosis of such cases presents no difficulty.

The venous stasis caused by cardiac failure may be complicated by thrombosis in the axillary and subclavian veins (Welch, 1900; Veal and Hussey, 1943). It is frequently a terminal event and in all such cases the cardiac disease has been advanced and the diagnosis was clear.

COURSE AND COMPLICATIONS

Partial and sometimes nearly complete recovery from the initial symptoms practically always occurs in a few days or in a few weeks whatever the treatment adopted.

Fatal embolism has not been reported. Maure (1914) gave an account of a woman of 71 years who fell and sustained a fractured humerus. She developed signs of venous obstruction in the same arm and died on the fourth day after the injury. Autopsy revealed a pulmonary embolism which originated from a thrombus in the axillary vein. The violence and nature of the injury excludes this case from the syndrome under consideration. It is open to question, moreover, whether a non-fatal embolism has occurred. Schepelmann (1911) reported its occurrence after phlebotomy and removal of a clot in a man with mitral valve disease and bradycardia. Roelsen's case (1944) suffered a reaction after the administration of heparin, and

the symptoms were believed in part to be due to a pulmonary embolism.

Involvement of the opposite arm is such an unusual event that the patient can be reassured. Only five such instances have been reported (Firth and Mackay, 1932; Selim, 1933; Taylor, 1933; Belilios and Houghton, 1935; Kux, 1935).

Despite the optimistic assertions by some authors, there is a strong tendency for symptoms to persist or recur. Matas (1934), Veal (1940), Roelsen (1939, 1944), and other writers who have made a special study of the condition have found that many

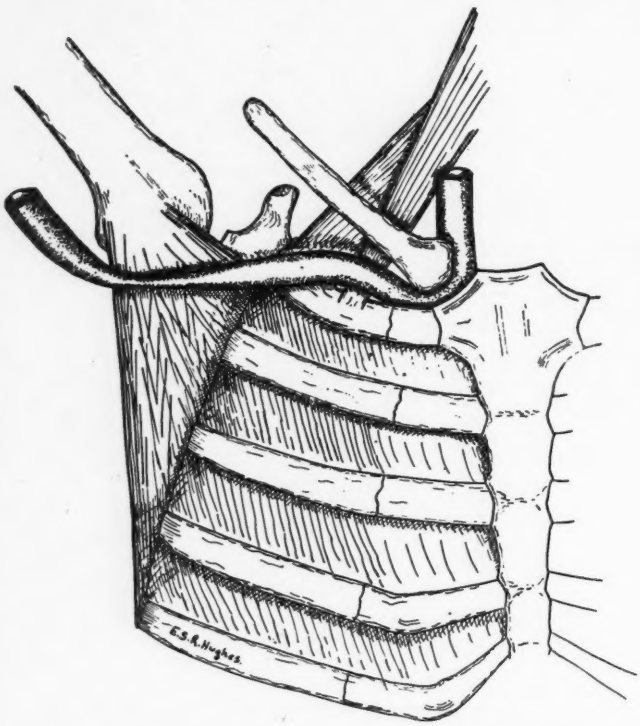

FIG. 217.—Veal and McFetridge (1935) believe that on abduction of the arm, the head of the humerus acting through the subscapularis muscle constricts and damages the axillary vein.

of the patients fail to make a complete recovery, or develop a recurrence of swelling, discoloration, venous congestion, and pain on exertion which renders them unfit to continue in their former occupations.

AETIOLOGY

Thrombosis of the axillary or subclavian veins has usually been regarded as the cause of the venous stasis. Ditter and Walker (1944) believed that insufficient attention had been paid to its spontaneous occurrence, but most authors have sought some predisposing cause. No chemical nor cytological abnormality has been found in the blood and no evidence of general or localized venous stasis. Since infection, either specific (Wilson, 1922) or non-specific (Lenormant, 1912; Lecene 1921; Chifoliau and Folliasson, 1932) cannot be regarded as anything but a very occasional cause of intimal injury in these cases, most attention has been concentrated on locating a vulnerable area along the course of the axillary and subclavian veins which might be traumatized in movements of the shoulder girdle.

Veal and McFetridge (1935) believed that in abduction of the arm, the head of the humerus acting through the subscapularis muscle constricted the axillary vein (*Fig.* 217).

Wright (1945) also suggested that hyperabduction of the arm might injure the vein; but he believed that the vessel was damaged as it passed posterior to the pectoralis minor just beneath the coracoid process (*Fig.* 218).

Lowenstein (1927) was the first to make a thorough anatomical investigation and he suggested that the vein was injured as it passed through the

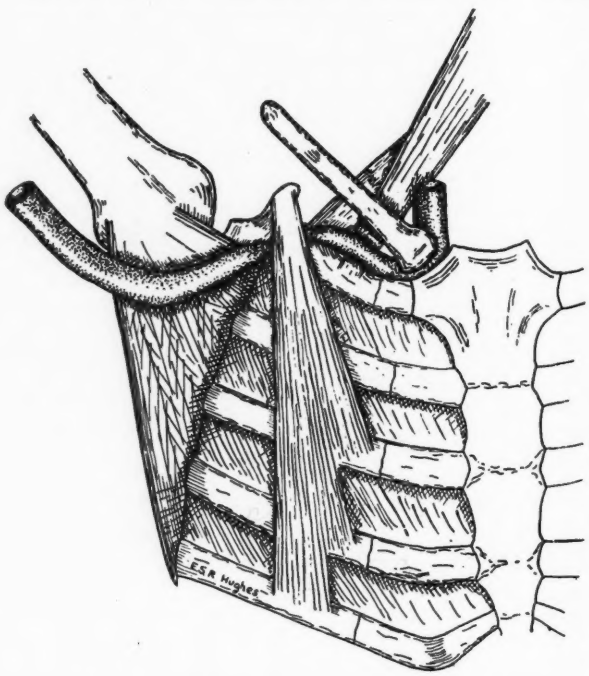

FIG. 218.—Wright (1945) suggested that in hyperabduction the axillary vein may undergo 'torsion, stretching, and pinching' as it passes posterior to pectoralis minor just beneath the coracoid process.

superior axillary hiatus. He believed that on abduction of the arm, the sharp crescentic lower border of the costocoracoid ligament pinched the vein against the rib. Gould and Patey (1928) injected liquid plaster into the axillary vein and were able to demonstrate on the plaster cast, of some of the individuals, a groove caused by pressure of the subclavius muscle or the costocoracoid ligament. They considered that such compression ruptured the subclavio-axillary valve, which is usually present in this situation. However, examination of intact autopsy specimens shows that on abduction the clavipectoral fascia, of which the costocoracoid ligament is a specialized part, is stretched as the coracoid process carrying the insertion of the pectoralis minor rotates posteriorly. This draws the ligament away from the rib and protects the vein from any localized pinching (*Fig.* 219).

Taylor (1933), Falconer and Weddell (1943) and Judovitch, Bates, and Drayton (1944) all believe that the subclavian vein may be compressed between the clavicle and first rib. There is no question that the

radial pulse can be obliterated by strong backward and downward bracing of the shoulders and this happens more easily when the neck is hyperextended. But the vein is protected in such a manœuvre by its mobility; it comes to lie between the subclavius muscle and the rib and such pressure as is caused by these two structures is unlikely to result in thrombosis. Moreover a cervical rib which, theoretically, might be a predisposing factor in such circumstances is rarely present (*Fig.* 220).

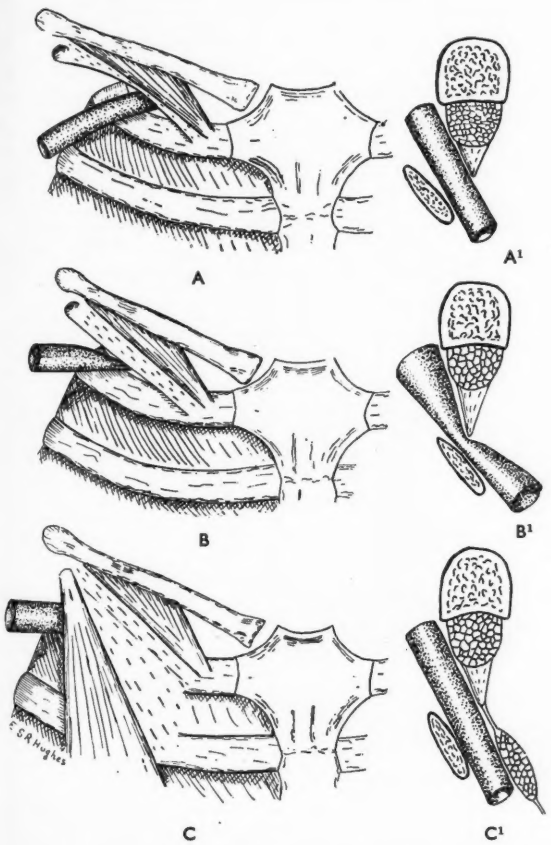

FIG. 219.—Lowenstein (1927) and Gould and Patey (1928) believed that the vein was damaged by the subclavius muscle or the costocoracoid ligament (A, A¹). If the clavipectoral membrane and pectoralis minor muscle be removed and the arm abducted, these structures, particularly the costocoracoid ligament, may compress the vein against the rib (B, B¹). But in the intact specimen this is prevented by the contraction of the pectoralis minor and the tension in the clavipectoral fascia which pulls the costocoracoid ligament away from the vein (C, C¹).

Sampson, Saunders, and Capp (1940) believe that the vein is traumatized in its course between the clavicle and the medial border of scalenus anterior. They suggest that an additional factor is stretching of the vessel, which is fixed proximally and dragged distally over the rib. But the mobility of the subclavian vein which causes it to slide beneath the clavicle has been observed at operation and can be demonstrated on the fresh cadaver (*Fig.* 221).

There are certain peculiar features in this syndrome for which the foregoing theories cannot offer an adequate explanation. It is impossible from an analysis of the movements of the shoulder girdle in these patients to select any single action as the main one responsible. It has occurred as a sequel to painting a ceiling (Gould and Patey, 1928) or to lifting heavy weights (Roelsen, 1944); in golfers (Gould and Patey, 1928) and in tennis players (Sampson, 1943); in a man pushing (Cottalorda, 1932) and in another pulling (O'Regan, 1941); the onset has followed an epileptic fit (Willcutts and Shelburne, 1943), and after a night's sleep (Ross, 1933).

If the lesion be primarily a torn intima acting as a focus for thrombus formation, one might expect

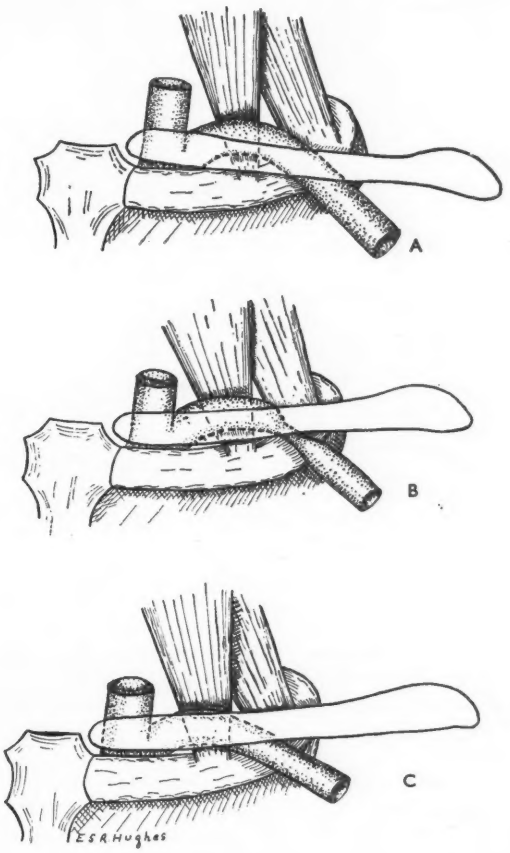

FIG. 220.—Taylor (1933), Judovitch et al. (1944), and Falconer and Weddell (1943) believe that the subclavian vein is compressed between the rib and the clavicle in certain movements of the shoulder girdle (A, B). But the mobility of the vein allows it to slip beneath the clavicle into a protected position (C).

a thrombus always to be found when surgical exploration is undertaken. But this has not been so (Wulsten, 1931; Ljunggren, 1935; Puhl, 1937; Hauge, 1942; and others). The most thorough exploration of the axillary and second and third parts of the subclavian veins that has been undertaken in this condition (in an operation by Dahl-Iversen on a case of Roelsen, 1944) revealed no abnormality of the veins. Moreover, in such a mechanism, the absence of any embolic phenomenon, or at the most its extreme rarity, is difficult to explain.

Yet the marked tendency for some degree of disability to persist suggests that the obstruction is nearly always a permanent one; the residual symptoms can be readily explained by the efficiency of the collateral vessels.

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INVESTIGATION

A study of twenty fresh autopsy specimens was made. The skin-flaps were dissected off the chest wall as far as the upper arm, and the axillary and subclavian veins were dissected to display their whole course, including their branches. The relation of the main vein to the structures of the axilla and neck was noted as were the alterations in the lumen of the vein produced by various movements.

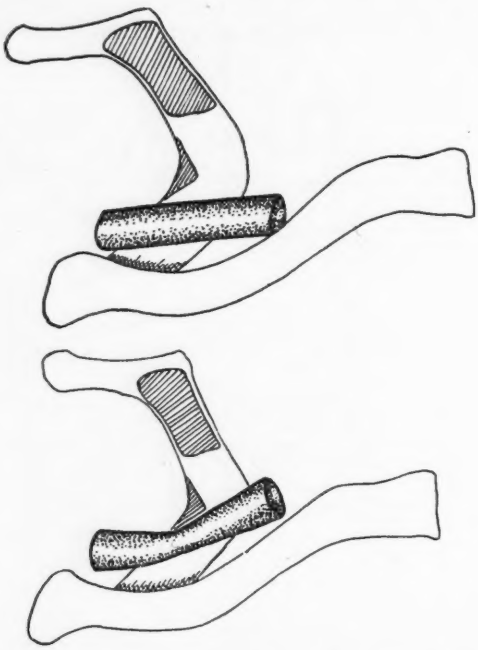

FIG. 221.—Sampson, Saunders, and Capp (1940) believe that the vein is injured between the medial border of the scalenus anterior and the clavicle. Since the subclavian vein is fixed proximally, they suggest that a further factor may be the drawing of the vein over the scalenus anterior tendon and first rib with certain backward movements of the shoulder girdle.

The axillary vein commences just proximal to the lower border of teres major, by the union of the basilic and brachial veins. It crosses the latissimus dorsi muscle and may come into contact with the capsule of the shoulder-joint before passing on to the subscapularis muscle. It then lies on the 1st and 2nd digitations of the serratus anterior and at the outer border of the first rib becomes the subclavian vein. This vessel, closely related to the first rib, courses forwards as well as medially, to cross the tendinous insertion of the scalenus anterior, and just medial to the medial border of this muscle it joins the internal jugular vein to form the innominate vein, which passes through the superior thoracic inlet into the superior mediastinum. The axillary vein for the most part lies deep to the pectoralis major and is crossed by the pectoralis minor and is covered by the clavipectoral fascia. At the superior axillary hiatus the vein is crossed by the costocoracoid ligament and subclavius muscle and clavicle, whilst in the posterior triangle of the neck the subclavian vein lies deep behind the clavicle and subclavius muscle.

The axillary vein, at first of narrow calibre, receives the small anterior circumflex vein; it widens at its junction with the subscapular vein. This vessel, of considerable size, receives the posterior circumflex humeral vein and communicates with the lateral thoracic vein by a short wide vessel. The lateral thoracic and acromiothoracic veins enter the axillary vein at the lateral and medial

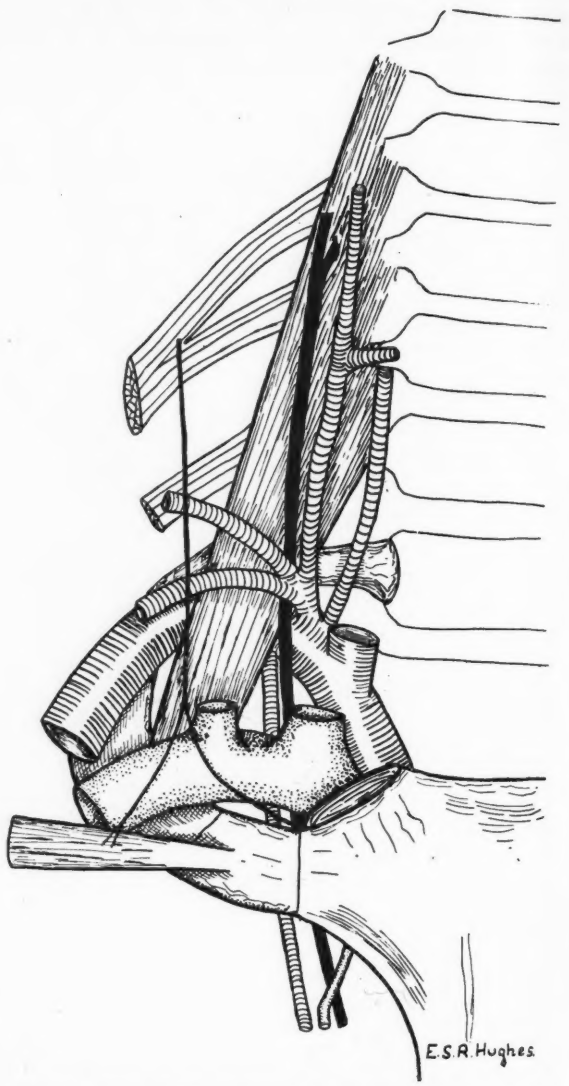

FIG. 222.—The phrenic nerve and the accessory phrenic nerve and their relation to the chief structures of the neck.

borders of the pectoralis minor respectively and between these two vessels a small, but constant, vein drains the deep surface of the pectoralis minor. The cephalic vein enters its first part after perforating the clavipectoral fascia. The external jugular vein drains into the subclavian vein as it crosses the scalenus anterior, and before doing so usually receives the suprascapular and transverse cervical vessels.

Movements of the shoulder girdle alone did not produce significant constriction of the vessel at any point in its course. In one specimen, depression of the diaphragm for 2 cm. caused complete obstruction of the vein, demonstrated by inability to force fluid into the innominate vein on that side. *In*

this specimen the phrenic nerve passed anterior to the subclavian vein. Normally, this nerve crosses the anterior surface of the scalenus anterior from its lateral to its medial border. It passes between the subclavian artery and vein, coursing from without in and slightly forwards. It rests on the sloping aspect of the pleural dome, lying in a vascular triangle formed by the subclavian vein above, the internal mammary artery laterally, and the internal mammary vein medially (*Fig.* 222). In the thorax the right phrenic nerve descends almost vertically with a slight anterior inclination; on the left side the nerve is practically vertical in the superior mediastinum, but curves around the heart, the concavity being directed anteriorly and medially, so that on this side the nerve is longer and it reaches the diaphragm farther from the midline than the right and at the same time on a more anterior plane.

The Aetiological Significance of a Prevenous Phrenic Nerve.—This abnormal position of the phrenic nerve has been described by several

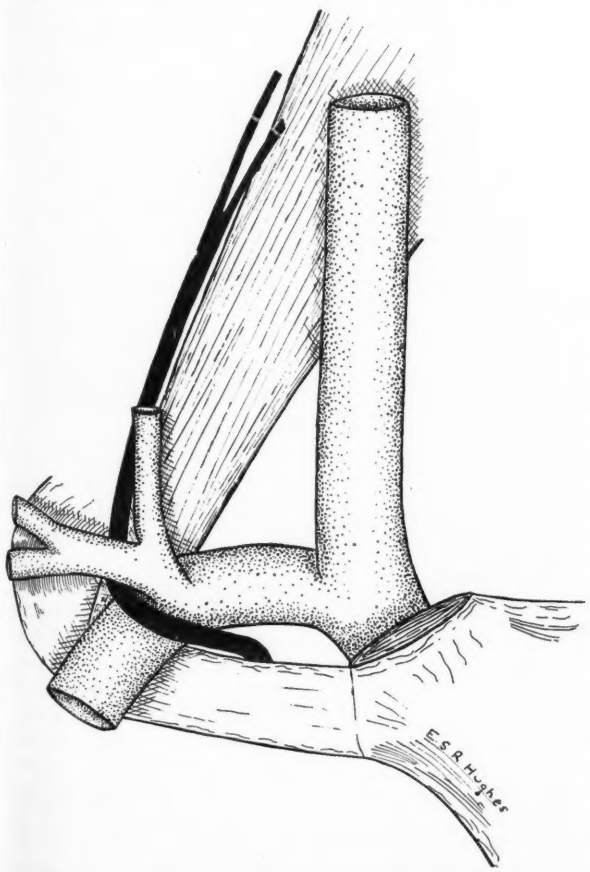

FIG. 223.—The prevenous phrenic nerve and its relation to the subclavian vein and tendon of the scalenus anterior. Note the lateral course pursued by the nerve. (Drawn from an actual specimen.)

anatomists. Hovelacque et al. (1936) found it in 10 out of 138 specimens, and in 8 of these all the filaments passing to the nerve coursed in front of the vein. They found that this prevenous nerve usually lies more lateral than the normal nerve and passes lateral to the venous tributaries of the angle of Pirogoff,* including the external jugular vein which is the most lateral of the tributaries (*Fig.* 223). Schroeder and Green (1911) found the abnormality in 4 per cent of cases and believe that it is not uncommon. In each of their cases the prevenous

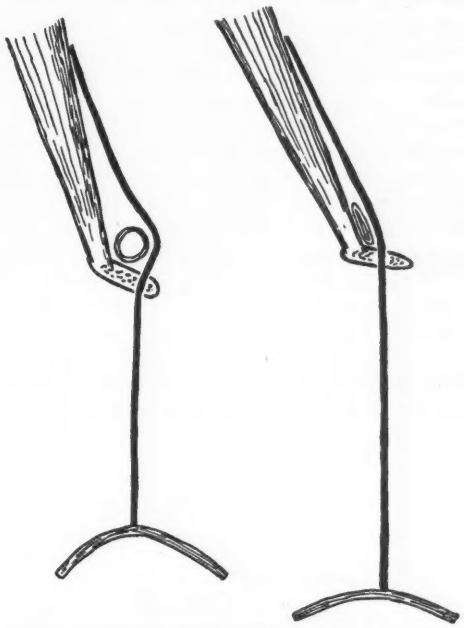

FIG. 224.—The prevenous phrenic nerve. On inspiration the descent of the diaphragm and the ascent of the first rib result in 'ligation' of the subclavian vein by the nerve.

nerve was on the right side, and *in one case the nerve actually coursed through the wall of the vein.* In one such case Wrisberg and Krause (quoted by Hovelacque, 1936) found that only intima separated the nerve from the blood as it passed through the wall of the vein. Rajanna (1947) noted that the accessory phrenic nerve might also occupy this intimate relation to the wall of the subclavian vein. These abnormalities receive ready explanation embryologically; the precursor of the main vein is a plexus of veins and usually that part of the plexus in front of the phrenic nerve develops into the subclavian vein, but this may be reversed, so forming the prevenous nerve; or the nerve may remain in intimate relation with the plexus to form a transvenous nerve.

The phrenic nerve is a tissue of considerable strength. Its abnormal anterior and lateral position in the base of the neck results in an increased angulation of the nerve as it passes into the thorax. In such circumstances the subclavian vein, which is crossed by the nerve as it lies on the tendinous insertion of the scalenus anterior and which in this part of its course is directed forwards as well as medially, is in potential danger of becoming constricted. This danger is increased in several ways.

Halls Dally (1908) has demonstrated that on quiet respiration there is little difference in the range of movement of the two halves of the diaphragm, but with deeper breathing the *right* dome moves more

* Pirogoff's angle: the angle formed by the internal jugular and subclavian veins (Dobson, 1946).

than the left. Further, the range of movement in *males* is considerably greater than in females. Again, during respiration the scalenus anterior contracts and elevates the first rib. Halls Dally (1908), in his study of 100 healthy patients between the ages of 15 and 35, found this movement to be more marked on the right side. Contraction of the scalenus anterior tightens its tendon, carries the subclavian vein farther forward, and increases the angulation of the prevenous phrenic nerve. When these respiratory movements are considered in conjunction with the shorter, more direct, course of the right phrenic nerve an explanation is forthcoming for the predominant involvement of the right arm. The importance of effort has been stressed by most, but its significance becomes more apparent when considered in terms of respiratory movements. Moreover, deep breathing during sleep is not at all uncommon. Extension of the neck increases angulation of the prevenous phrenic nerve (*Fig.* 224) and thus probably increases the possibility of subclavian vein obstruction. It is an essential attitude when performing overhead work; it is frequently assumed when lifting heavy weights or whilst sleeping.

This theory of prevenous phrenic nerve obstruction offers an adequate explanation of an apparently permanent obstruction. The nerve constricts the vein in the circumstances as efficiently as a ligature. Recovery depends on the efficiency of the collateral circulation, which in turn will depend on whether the prevenous nerve passes medial or lateral to the external jugular vein, and the extent of secondary thrombus formation, should this occur.

Such an obstruction so close to the jugular vein can rarely result in embolism; moreover, the site, hidden deep to the sternomastoid and clavicle, is most inaccessible in the usual operative exploration.

TREATMENT

In most cases conservative treatment has been followed from the outset and many such patients have made a rapid and apparently complete functional recovery. On the other hand other cases have run a protracted course and in such circumstances the surgeon has intervened. Various surgical procedures have been followed: phlebotomy, with removal of a thrombus; excision of a thrombosed venous segment, with or without periarterial sympathectomy; perivenous sympathectomy; and venolysis—all have been tried with varying success.

McLaughlin and Popma (1939) reported an excellent result following scalenotomy.

Anticoagulants should be used from the outset. Thrombosis, which might complicate any form of obstruction, tends to propagate, so blocking important collateral vessels. Heparin and dicumarol reduce this tendency and if used after the onset are a most valuable adjunct to treatment. (*Fig.* 225.)

Paravertebral cervical sympathetic blocks have also proved a valuable aid to recovery (de Bakey, Ochsner, and Smith, 1942; MacCarthy, 1946; Learmonth, 1948). The procedure interrupts impulses originating from the affected segment which might pass centrally and reflexly affect the peripheral circulation, and it has probably a more important effect in removing vasoconstrictor impulses from the collateral vessels (*Fig.* 226).

Such a line of treatment followed promptly will probably render any surgical treatment unnecessary, though a phrenic crush which would temporarily hinder the diaphragmatic movements might be considered.

In those cases where disability remains some years after the onset, a venous anastomosis is indicated, utilizing the external jugular, anterior jugular, or even the internal jugular.

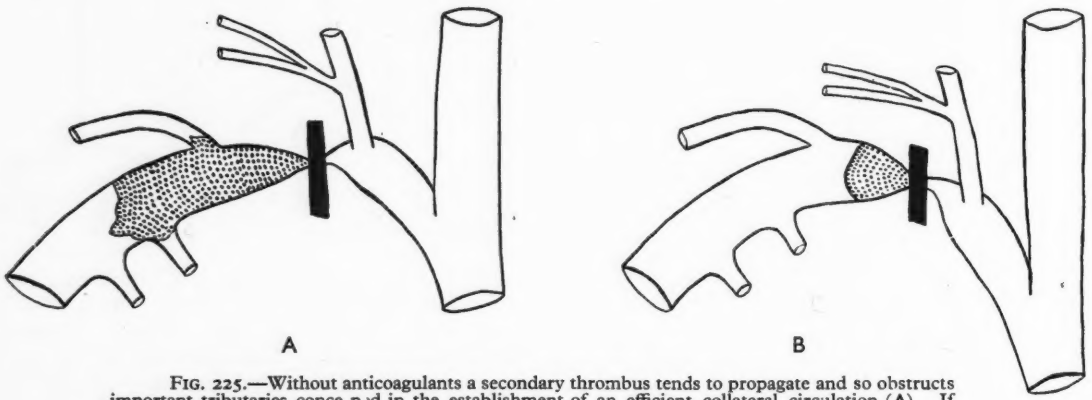

FIG. 225.—Without anticoagulants a secondary thrombus tends to propagate and so obstructs important tributaries conce n :d in the establishment of an efficient collateral circulation (A). If anticoagulants be used a secondary thrombus, if it forms, is prevented from spreading (B).

SUMMARY

Venous obstruction with a comparatively sudden onset may occur in the upper extremity of an otherwise healthy person. It is characterized by swelling, discoloration, and distension of the superficial veins and the arm is sometimes painful. The prognosis, so far as life is concerned, is excellent, but residual after-effects, varying in degree from trivial to serious, persist probably in all cases, suggesting that obstruction is permanent with recovery dependent upon the efficiency of the collateral vessels.

Many explanations have been given to account for the obstruction, but all these depend ultimately on the formation of a thrombus in the axillary or subclavian vein. In actual fact, however, operation has often disclosed no sign of a thrombus. The research recorded here indicates that a phrenic

nerve which passes anterior to the subclavian vein, a prevenous phrenic nerve, may constrict the vessel against the tendon of scalenus anterior, as effectively as a ligature.

Anticoagulants should be administered as soon as possible to limit the extent of intravascular clotting which may follow secondarily and which

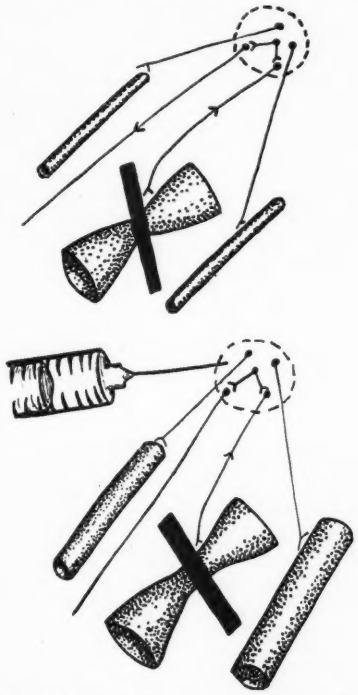

FIG. 226.—Injection of the stellate ganglion with novocain exerts a favourable effect on the rate of recovery. This is probably largely due to dilatation of the collateral vessels and, to a lesser degree, lifting vasoconstrictor impulses from the peripheral circulation.

may obstruct vessels concerned in establishing a collateral circulation. Paravertebral sympathetic blocks have shortened the convalescence, probably by their influence on the collateral vessels.

I would like to thank my senior colleagues, Mr. A. Cameron MacLeod, Mr. J. Thompson Fathi, and Mr. Charles Gray, for referring certain of these patients to my clinic, and Professor F. Wood Jones for his stimulating advice during the preparation of the paper.

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IMPLANTATION OF SECONDARIES FROM A RENAL CARCINOMA ('HYPERNEPHROMA') WITHIN THE URETERIC LUMEN

By JAS. B. MACALPINE, Manchester

This communication reports briefly the recurrence of a renal carcinoma on the ureteric wall.

CASE REPORT

Mrs. M. M., aged 39, attended the urological department of the Salford Royal Hospital in September, 1932, complaining of periodic attacks of difficulty in micturition associated with the passage of blood and clots. An aching pain was present in the left loin.

On Examination.—Abdominal examination revealed a large mass in the left hypochondrium having the characteristics of a renal tumour. The urine was heavily blood-stained. A radiograph was negative and radiology of the

Fig. 227.—The specimen, showing the two lesions.

chest and limbs discovered no secondaries. The cystoscopy revealed a healthy bladder, except for trigonal œdema. From the left ureteric orifice issued what looked like pure blood. Indigo-carmine appeared on the right side in 4½ min., but was absent from the left during the eleven minutes it was under inspection. A pyelogram showed the ureter pushed inwards so as to overlie the vertebral column. The opaque solution diffused itself somewhat widely, giving an ill-defined, characterless shadow and not outlining any calices. The renal outline was large. Hæmoglobin 43 per cent. Transfusion.

At Operation.—Nephrectomy. The operation was one of unusual difficulty owing to the size of the tumour and to dense adhesions, especially at the upper pole. The last rib had to be removed. The growth extended about 2 in. into the renal vein.

Progress.—The prognosis was regarded as unfavourable. Nevertheless, the patient was well till January, 1937, and had put on considerable weight. She then noticed small clots in the urine which, however, was never bright red, and she had some suprapubic pain. The abdominal and vaginal examination and radiology revealed no abnormality. At cystoscopy (April 22, 1937) the left ureteric orifice was seen to move as for an efflux, and a faintly coloured efflux was suspected. Pressure and gentle kneading over the left renal region and the iliac fossa produced a well-marked, blood-stained issue. A ureterogram was unhelpful. Radiography of chest, etc., negative for secondaries.

Second Operation (May 21).—Ureterectomy. The upper end of the ureter was obviously distended and

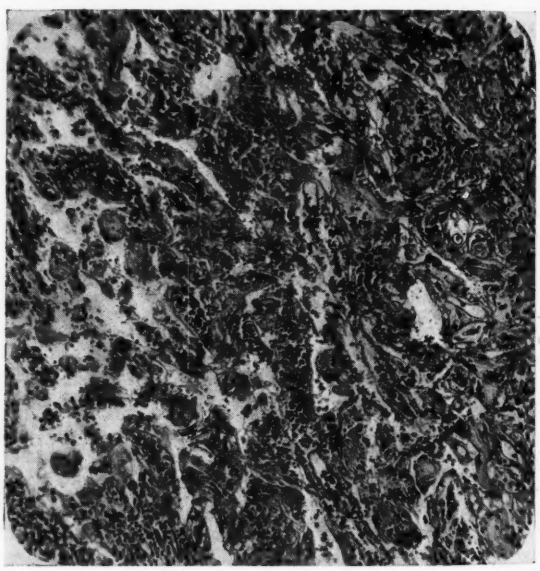

Fig. 228.—Section showing tissue typical of renal carcinoma.

pathological. The tube was removed to the juxta-vesical area. The specimen (*Fig.* 227) shows two lesions: (*a*) an elongated, fleshy, polypoid mass, springing by a narrow pedicle from just below the upper closed end of the ureter, the wall of which shows some puckering at the site of origin. The tumour has a deep purplish-red colour and is mottled. It has a smooth surface and is about 3 in. long. (*b*) At a point corresponding to the junction of the middle and lower thirds of the ureter a second lesion is observed, about the size of a match head and sessile, but otherwise of appearance similar to (*a*). Sections of (*a*) and (*b*) both showed tissue typical of a renal carcinoma (*Fig.* 228).

May 9, 1945: Returned to medical out-patients department with frontal and left-sided headaches, some loss of memory and incoherence, diplopia, etc. Transferred to Professor G. Jefferson who removed a metastasis from the left temporal lobe which, on section, showed

a "secondary hypernephroma". She made a good recovery from this, but died in May, 1946, with deposits in the liver, etc.

DISCUSSION

The renal tumour removed in 1932, though bulging somewhat into the pelvis in the usual manner, did not, as sometimes, extend into the ureter, and in the author's opinion the higher of the two ureteric tumours could not have been implanted by direct contact or friction with the parent tumour. This is emphatically true of the lower growth, which would have been quite out of reach. In both cases it appears probable that a detached fragment of tumour has adhered to the mucosa where it has engrafted itself. The lower tumour is so small that it is hard to believe that it can have been a graft from the original renal growth removed nearly five years previously and it is suggested that it may be the offspring of the larger ureteric neoplasm—a grandchild of the kidney tumour.

This is the sole example in the author's practice where a hypernephroma has produced a secondary implant on the ureteric wall and he cannot recall having seen any specimen or case in the practices of others. A similar phenomenon is, of course, well known in papillomatous disease and of these latter the author has seen a number of examples.

From urological and other monographs the following quotations are taken :—

Albarran and Imbert make the general statement that, "In certain neoplasms of the kidney which have invaded the pelvis the cavity contains . . . cancerous nodules which are not in direct continuity with the neoplasm : these may be considered actual neoplastic grafts on the wall of the pelvis which is in contact with the neoplasm". Reference is then made to the cases of Israel and Pasteau (*see below*).

Israel says : "Though it is principally the papillomatous tumours which cause grafts in the ureter and bladder, the tumours of the renal parenchyma are also capable of producing such grafts."

Legueu says: "The tumour . . . quite often extends to the pelvis and to the upper extremity of the ureter ; at other times the propagation occurs by grafting at a distance on these conduits, either through contact of the tumour with the pelvic wall (Pasteau), or by means of the urine at the inferior opening of the ureter."

Young : "The growth may extend down the ureter, even to the bladder, with or without involvement of the ureteral walls."

Marion : "Propagation by graft to the pelvis or ureter is altogether exceptional."

Bland-Sutton : "The cancerous tissue creeps into the pelvis of the kidney and invades the ureter, extending sometimes the whole length of the duct, and the outrunner has been observed to enter the bladder."

These vague pronouncements, for the most part unsupported by reference to specific examples, suggest to the writer that from early days renal carcinomas may have been confounded with papillomas, and copying from text-book to text-book may be responsible for over-emphasizing the prevalence of a phenomenon which, though actually happening, is very exceptional.

A painstaking search of the literature has been made for *authentic examples* of grafting downstream from renal carcinomata and this search only emphasizes their extreme rarity.

Pasteau (1902) showed a kidney which had been removed for a "cancer". The tumour had put out a prolongation the size of a nut which filled the pelvis, and had contracted secondary adhesions to the internal surface at two points removed from the true base of origin.

Richards (1905) recorded the case of a man aged 58 who died in 1878. At post-mortem examination "a large, firm, infiltrating growth occupied the upper half" of the right kidney and involved the adrenal and liver. The pelvis was "dilated and sacculated. The ureter was dilated and thickened and over its upper half its mucous membrane is covered by a vascular growth from its surface. This seemed to have begun half-way down the canal, and growing thence had pushed upwards as a clot might do, forming a mould of the ureter at the upper part and merely attached below . . . Microscopically : large round cells, some of them rather like epithelium —'a medullary cancer rather than a sarcoma'." The large round cells, the mould of the ureter, and other features suggest that this is a true example of a renal carcinoma implanted in the ureter.

Seizo-Suzuki, of Tokio (1909), reported a male patient aged 43 who suffered from hæmaturia. Blood was seen coming from the left ureteric orifice in quantity. Nephrectomy was refused. Death supervened from cachexia. Post mortem, the left kidney contained a tumour the size of two fists involving the suprarenal and the pancreas. On the lower edge of the left ureteric orifice, which was somewhat dilated, sat a whitish, mushroom-shaped tumour about the size of two hemp seeds. Its pedicle was short and about 1 mm. in diameter. The author labels the renal tumour "a neoplasm of the left suprarenal penetrating the left kidney". It consisted of epithelial cells and the nuclei stained well. The cells were of many sizes and shapes, the largest being approximately ten times the size of a normal kidney cell and the smallest also larger than a kidney cell . . . The bladder tumour was structurally identical with the renal one. The tumour mass in the bladder can, in Suzuki's opinion, only be explained by implantation. The primary tumour was in the present writer's view a renal carcinoma.

Cahn (1911) discussed the case of a 59-year old man who died shortly after operation. The kidney contained a "hypernephroma" and the ureter was distended to an average breadth of 3 cm. Springing from its wall were isolated tumours which histologically were shown to be "hypernephromatous" in structure. Lung secondaries were also found. Cahn says he can find only 3 comparable cases in the literature, all American, but gives no reference. He appears, without justification, to regard the secondary as a metastasis rather than a graft.

Pleschner (1913) contributes a paper detailing 25 case histories of "hypernephromata". In No. 15 of the series a renal tumour of large size was removed from a man aged 55. Part of the renal tumour almost the size of an egg protruded into the pelvis, completely filling it. The patient died and at post-mortem the proximal 3 cm. of the ureter were

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distended to the size of a finger, the obstruction being caused by a tumour the size of a bean growing from the ureteric wall. Below this point the ureter was of normal size. Pleschner remarks that this is the rarest method of dissemination for hypernephromata.

Haebler (1920), at a session of the Berliner Urologischen Gesellschaft, demonstrated an implantation metastasis from a "hypernephroma", the patient returning three years after nephrectomy with renewed hæmorrhage. The tumour was seen cystoscopically in the region of the left ureter. It was the size of a walnut and projected through the severely dilated orifice. The ureter was removed, together with the surrounding circle of the bladder.

Dobrotworski (1921) reported the case of a man aged 44 who was nephrectomized for a "typical hypernephroma" which, though small, reached into the upper calix. Some 4 cm. of healthy ureter was removed with the kidney. Within four months bleeding started again and subsequently recurred periodically. After 3½ years the ureteric stump was removed and was found to contain a compact, sausage-like, pedicled tumour about 1 cm. in length which was dilating and blocking the lumen. It was situated 5 cm. below the renal pelvis. Its microscopical structure was identical with that of the renal tumour and "as the intervening ureter was healthy it must have been an implantation". Moreover, the metastasis was not mucosa-covered. Dobrotworski operated upon this patient in Professor Federoff's Klinik in St. Petersburg and says there are three specimens of the condition in the museum of that service. The same case is mentioned by Federoff himself in an article on the treatment of renal tumours. Dobrotworski mentions cases by Clairmont and Watson which have not been traced.

Thomson-Walker (1921) reported 4 cases of growth of the ureter. The first three were papillomata. The fourth was a man who had had a nephrectomy on Aug. 10, 1920. "Kidney showed a hypernephroma, round in shape and embedded in the upper half of the kidney. Renal pelvis distended with blood-clot but otherwise normal." On Sept. 28, hæmaturia recurred and on Nov. 14 he was readmitted with severe hæmaturia which had appeared four weeks after leaving the hospital. Ureterectomy. "The specimen of the left ureter showed a small growth about the size of a split pea at the upper end, and two blood-clots along its course." Thomson-Walker makes no comment on this case and reports no section either of the original tumour or of the secondary in the ureter.

Hunt (1930) narrates 2 cases in which "involvement of the ureter, secondary to the primary renal lesion was found." Man aged 47, left nephrectomy (Nov. 29, 1922) by C. H. Mayo. Kidney "was classified pathologically as hypernephroma". Nearly five years later hæmaturia which was traced to the ureteric stump. Left ureterectomy (Dec. 16, 1927). "Found to be an epithelioma graded 2." Three months later cerebral symptoms supervened. At a decompression operation "a specimen from the tumour was removed for diagnosis. It proved to be a metastatic epithelioma".

Man, aged 44, diagnosed as left renal tumour. "Aug. 5, 1927, left nephrectomy was done for a large tumour of the kidney which was classified as hypernephroma." Patient returned March, 1928, on account of hæmaturia. Traced to stump of left ureter. Ureterectomy. ". . . the upper portion was involved by invasion of the hypernephroma."

Neither of these cases is completely free from doubt. In the first an epitheliomatous secondary, would, with greater probability, arise from a primary which also was an epithelioma and therefore from a pelvic growth. In the second the text does not make it clear whether the ureter was invaded from without by a local recurrence or whether it was a seedling implant on the ureteric wall transmitted with the urinary stream.

Schacht (1931) published the case of a man, aged 55, who had left nephrectomy at the Mayo Clinic for "a hæmorrhagic degenerating hypernephroma". Ten months later he had severe pain in the left side and hæmaturia. A ureterogram showed "that the ureter was not filled; about 1 cm. of the upper part was evidently considerably dilated". Cystoscopy: blood-clots in the bladder. The passage of a ureteric catheter caused considerable bleeding from the ureter. Ureterectomy, the tube being divided just below the brim of the pelvis. Pathological report: "A hæmorrhagic papillomatous hypernephroma (2 × 1 cm.) was found attached to the wall of the ureter".

Szymonowicz (1932) reports a 56-year old woman in whom a tumour of the left kidney was diagnosed by means of a pneumoperitoneum. Simultaneously a small, ulcerated tumour close to the external meatus was observed, which on microscopical investigation proved to be an "atypical malignant hypernephroma". At cystoscopy a tumour was seen situated at the right ureteric orifice which was the size of a hen's egg and bled on being touched. Szymonowicz regards the case as worthy of remark because the simultaneous tumours of the bladder and vulva led to the presumption that they arose through direct implantation of the "hypernephroma" via the urinary passages.

Cibert and Perrin (1938) recorded the case of a male, aged 47, who had symptoms in 1931 followed in 1933 by nephrectomy. A very difficult operation. "Hypernephroma." Sept. 13, 1935, renewed hæmaturia, total, important. Cystoscopy—in the region of the left ureteric orifice a "*tuméfaction*" the size of a cherry, rounded, and covered by normal mucosa. Remainder of the bladder healthy. Removal of the lowest part of the ureter together with an area of the bladder surrounding the left ureteric orifice. The removed ureter contained a tumour in its intramural portions which looked like a clot in process of organization and was quite soft. The specimen was purposely sent to the laboratory without a history and proved puzzling, being at first considered of "*type allantoïdien*". The clinical history was then supplied and the report was changed to "*épithélioma renal à cellules claires comme la tumeur primitive*". Subsequent history: Shortly after leaving the hospital the patient developed headaches and vertigo, and about a year later a decompression operation was carried out with the diagnosis of a cerebral metastasis. Death in the summer of 1937, i.e., six years after

the appearance of his first symptoms and four years after the nephrectomy.

It is noted that, like my own patient, this man ultimately died from a cerebral metastasis, as did one of Hunt's two cases.

Doubtful or Spurious Cases.—Amongst the cases above reported some are undoubted examples of implantation metastasis to the lower urinary passages; others are less convincing, either through inadequate detail or because they belong to a period before the modern classification of renal tumours, and their precise nature is difficult to decide. Whilst accepting the evidence for himself the author realizes that some readers may reject one or more cases. The following are spurious.

Israel (1901) mentions a case in which a vascular tumour the size of a pea occurred at the ureteric orifice. Its type corresponded with that of a tumour found in the kidney. The kidney was replaced by a firm yellowish-white neoplasm. The pelvic wall also showed diffuse neoplastic degeneration and on its mucosa were spongy, tufted (*zottige*) excrescences. No microscopical examination is appended. This has been referred to by Albarren and Imbert and others as a parenchymal tumour, but, in view of the description, the present writer inclines to the opinion that the growth was a papillo-carcinoma primary in the pelvis which had secondarily invaded the renal parenchyma and had developed a graft to the lower parts, a process which, of course, is much commoner with pelvic than with parenchymal growths.

Hoffmann (1907) has reported a case of bladder involvement by a hypernephroma, but it appears to come into a different category as a large secondary growth had developed in the true bony pelvis and the pelvic organs were embedded therein. It had perforated the anterior and posterior vaginal walls and presented in the lumen. It had also perforated the roof of the bladder where two tumour masses were found. The diagnosis was made by sectioning a secondary which had appeared on the thigh. The hypernephromatous nature of this led to a cystoscopy. The renal tumour was confirmed post mortem. Though this is a genuine case of involvement of the bladder by a growth secondary to a renal carcinoma, the route of transference is evidently not by the urinary stream.

Bissell (1914): A male, aged 58, died of uræmia. At the post-mortem there was a renal carcinoma of the upper pole which extended into the pelvis of the kidney and down the ureter—very nearly to the bladder—also into the mouth of the renal vein. . . The ureter was greatly distended (2·5 cm. in diameter) and its lining was made up of friable tumour tissue, 1 cm. in thickness. . . The same tissue lined the pelvis of the kidney, was uniform in thickness throughout the pelvis and ureter, and ended abruptly 1 cm. above the ureteral orifice.

After reviewing current opinions regarding the lymphatics of the kidney and ureter and having remarked the absence of lymphatic involvement except in microscopical sections from the upper portion of the ureter, Bissell concluded that " because there is so little involvement in the lymph-nodes adjacent to the ureter, and because only in the uppermost, oldest portion of the tumour invasion of the ureter, is there demonstrable tumour tissue in lymph-channels, it seems highly probable that extension took place along the mucous membrane of the ureter, and after destruction of the mucosa and submucosa extension into the lymphatics of the muscle coat occurred ".

It is questionable whether what appears to be not an implantation from the urinary stream but a direct spread along the mucosa is strictly parallel with the case now reported.

COMMENTARY

The cases collected above have been discovered only after an extensive search of the literature and their marked infrequency supports the view that implantation in the ureter is a very exceptional phenomenon, though definitely established. Quotations taken from standard text-books leave one with the impression that it is a complication to be met with now and again. It is suggested that, of old, examples of dissemination of papilliferous material to the ureter from the pelvis were misdiagnosed as " hypernephromatous " and the faulty idea thus obtained of the latter's frequency has been perpetuated through the statements of earlier writers being accepted uncritically.

It will be noticed that my patient, who had an unusually large renal carcinoma which invaded the renal vein and was almost inoperable, survived from September, 1932, to May, 1945 (nearly 13 years), before a distant metastasis revealed itself. This length of survival is unusual.

Garceau (1909) examined the results of 143 nephrectomies for renal growths. There was an operative mortality of 23 per cent (33 cases), and 36 could not be traced. Of the remaining 74 patients, 33 died in the first two years (44 per cent) and there was only 1 survivor at the end of the tenth year. The average expectation of life was 3½ years, dating from the first symptom, and only 7·10 per cent of patients survived 4 years.

Among 193 patients traced by Hand and Broders, of the Mayo Clinic, (1932) 23 died in hospital, 50 lived less than one year, 89 died during the first two years; 24 lived ten years or more and in 5 of these latter extension to the renal vein had taken place.

Jocelyn Swan (1933) collected 51 cases of renal tumours, of which only 32 were operable when first seen. He traced the 32 cases which had been operated on. Only 8 (25 per cent) were alive at the end of three years and only 1 after ten years.

Ross Mintz (1937) reviewed 105 consecutive cases of renal cancer, of which 72 had been nephrectomized; 45 of them were dead, and of the 22 survivors only 7 had exceeded a five-year period.

It is fascinating to speculate what had been happening to my patient's tumour during a period of thirteen years. When was the cerebral transplant detached from the parent growth? Or was it an offshoot from one of the ureteric growths (which is unlikely)? Had it lain within the brain all these thirteen years, and if so had its evolution been slow and uniform or had it been dormant for most of the time and suddenly sprung into activity? And do tumours within the kidney itself behave similarly, having an unsuspectedly long latent existence before ultimately disclosing their presence?

A case not irrelevant to the present discussion is reported by Bull, of Oslo, and provides food for

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further reflection on the behaviour of these tumours. A thrombus in the renal vein was included in the ligature. Microscopical examination of the portion removed showed it to be composed of tumour cells. At autopsy six years later not only were no metastases found, but there were no tumour cells in the renal vein.

An instance of an isolated metastasis is reported by Finsterer. A patient underwent nephrectomy in 1897. A secondary in the scapula was removed in 1903 and in 1913 the patient was free from recurrence.

Another interesting example is narrated by Dellinger Barney who nephrectomized a patient in 1932. A "solitary" metastasis in the lung was treated by removal of two-thirds of the upper lobe in 1933 and the patient was well in 1944 (twelve years after nephrectomy).

My sincere thanks are due to the library staff of the Royal Society of Medicine for invaluable help in searching the literature.

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MEGALO-URETER: A REPORT OF TWO CASES

BY J. H. CARVER, LONDON

TWO cases of megalo-ureter apparently due to neuromuscular imbalance at the uretero-vesical junction are reported.

CASE REPORTS

Case 1.—E. C., aged 29, was admitted to hospital on Feb. 23, 1946, with frequency and scalding of over one month's duration. She was a primipara and was six months pregnant. She had been treated with alkalis and albucid without effect.

ON EXAMINATION.—Tenderness in the right renal angle, kidney not palpable. B.P. 150/90. Urine: a few R.B.C.'s, numerous micro-organisms, no growth on culture. Blood: R.B.C. 4·2 million, Hb 74 per cent, C.I. 0·9, W.B.C. 13,200, Polymorphs 77 per cent, Lymphocytes 22 per cent, Monocytes 1 per cent, Albumin 0·03 per cent, Blood-urea 25 mg. per cent, Disks normal. X-ray examination of the heart: No cardiac enlargement. Plain radiograph: Right vertex presentation, head high; no opaque stones seen. I.V.P.: Left, slight pelvic and caliceal dilatation; Right, kidney enlarged, poor secretion of dye but sufficient to make a diagnosis of hydronephrosis (*Fig.* 229).

TREATMENT.—She was treated with sulphanilamide and alkalis. Her frequency diminished and her general condition improved: she was discharged on March 11. Repeated examination of the urine, however, revealed pus, but no organisms were found.

ON READMISSION.—She was readmitted on April 12 with recurrence of the symptoms. There was slight hydramnios and œdema of the ankles. B.P. was still 150/90 and blood-urea 21 mg. per cent. There was heavy deposit of pus in the urine, but cultures were sterile. She was transferred to the Obstetric Department of the Hammersmith Hospital and was confined there on April 28. Labour was normal. On May 12 an I.V.P. was done which showed the left kidney to be normal, but no dye was seen on the right side even after sixty minutes. The urine still contained pus and Gram-negative bacilli, with some diphtheroids. Cystoscopy revealed a mild trigonitis, the ureteric orifices were normal, and a full-sized catheter was easily passed up to the right kidney. A retrograde pyelogram showed an enormous right hydronephrosis and a widely dilated right ureter, the dilatation extending down to the bladder (*Fig.* 230). As she was nursing her baby, operation was postponed.

AT OPERATION.—She was operated on on Nov. 2, a nephro-ureterectomy being done. There were many dense perinephric adhesions, the ureter was dilated, thickened, and tortuous throughout its whole extent, particularly in the lower third. The kidney substance was almost completely destroyed (*Fig.* 231).

HISTOLOGY (Dr. C. V. Harrison).—Sections from the kidney have been taken through the whole thickness from capsule to pelvis and show gross hydronephrosis the total thickness of the kidney varying from 2 mm. to 4 mm. In spite of the great thinning, the majority of the kidney tissue included in the sections is well preserved and only odd foci show any destruction of renal tissue. In these foci the glomeruli have largely disappeared and the tubules are dilated and filled with a protein-containing fluid. The appearances suggest that these are foci of healed pyelonephrosis rather than simple hydronephrosis. The pelvis shows a partial loss of

epithelium and a localized zone about 200 to 300 μ thick of cellular infiltration consisting mainly of macrophages and plasma cells.

A transverse section of the ureter shows great dilatation, with an external diameter of 13 mm. and a thickness of 2 mm. (*Fig.* 232). The lining epithelium is intact and appears normal. The submucosa is denser than normal, consisting largely of well-formed collagen, and shows slight cellular infiltration, mainly by plasma cells. The muscularis is normal in its arrangement but appears hypertrophied.

The appearances as a whole indicate gross hydronephrosis and hydro-ureter associated with a mild infection.

FIG. 229.—Excretion urogram when six months pregnant very poor secretion on the right side, but enough to make a diagnosis of hydronephrosis.

FIG. 230.—Retrograde pyelogram. Hydronephrosis: the ureter is dilated and tortuous.

FIG. 231.—The specimen has shrunk owing to being placed in too strong a solution of preservative. The thickened ureteric wall and the narrow strip of renal parenchyma are seen.

FIG. 232.—Section of the ureteric wall. (× 4.)

Case 2.—W. G., aged 13, was knocked down by a cyclist on Dec. 28, 1946, and was admitted to hospital immediately afterwards. He was concussed but not unconscious, and no fractures were found on examination. He complained of severe headache and routine investigation revealed a blood-pressure of 220/120 with thickened radial arteries. The heart was not markedly enlarged and the disks, apart from fullness of the retinal veins, were normal.

Investigation: Urine, clear, albumin 1+, a few pus cells and red blood-cells, very occasional epithelial cells. Gram film: occasional Gram-positive cocci. Cultures sterile. Blood-urea 46 mg. per cent. Urine urea 1·08, 1·18, and 1·38. W.R. and Kahn negative. Blood-count: W.B.C. 10,000, Polymorphs 82 per cent, Lymphocytes 15 per cent, Monocytes 3 per cent. Cerebrospinal fluid: Clear, colourless; Protein 30 mg. per cent; Globulin very faint trace; Chlorides 650 mg. per cent, Cells 1 lymphocyte, 2 polymorphs. Urea-clearance: 74 per cent of normal. I.V.P: Owing to faulty preparation, no detail could be made out. Cystoscopy: bladder normal, ureteric orifices normal, catheter passed easily. Left kidney, normal pyelogram: right kidney, small, about half the usual size. Ureter greatly dilated (*Fig.* 233).

FIG. 233.—Retrograde pyelogram. Dilated tortuous ureter with small atrophic kidney.

OPERATION.—Nephro-ureterectomy: There were many dense perinephric adhesions: the ureter was dilated tortuous, and thickened, and was the size of the small, intestine just before it joined the bladder (*Fig.* 234).

AFTER-HISTORY.—Seven months later it was found that the blood-pressure was 120/80, and a family investigation revealed the father's B.P. 148/90, the mother's 140/86, and the sister's (aged 18) 110/60.

PATHOLOGY (Dr. I. Doniach).—Kidney 6·5 × 3·5 × 2·5 cm. Capsule stripped easily: subcapsular surface pale, showing deep coarse irregular scars but no fine granularity. Cut surface showed alternating scarred and fairly normal parenchyma (it is 1·5 cm. thick). The pelvis is a little dilated and its wall hypertrophied. The renal artery and branches are thick-walled and the lumen considerably reduced. Ureter 13 cm. long, very thick-walled, with a distended lumen, the distension increasing peripherally (the diameter just below the pelvis is 0·6 cm. and just above the ureterovesical junction 1·3 cm). The sections show wedge-shaped parenchymal ischæmic scars heavily infiltrated with lymphocytes alternating with fairly normal parenchyma. The latter shows occasional partially hyalinized (ischæmic) glomeruli but no tubular atrophy. In the ischæmic wedges hyalinized glomeruli and atrophic tubules are a prominent feature. Colloid-containing tubules lined by a flattened epithelium are common in the ischæmic areas. The pelvic submucosa is heavily infiltrated with lymphocytes. The interstitial tissue of the papillæ shows a marked fibrosis. The interlobular arteries all show a marked medial hypertrophy and intimal elastosis with a reduced lumen to wall diameter ratio. The afferent arterioles show hypertrophy but no hyalinization. The renal and arcuate arteries are hypertrophied but free from intimal thickening. The above appearances are certainly not those of a classical hydronephrosis. The picture now is one of patchy old ischæmic atrophy and a hypertensive hypertrophy of the small arteries. That the former lesion resulted from a congenital achalasia of the ureterovesical junction can only be presumed from the history rather than proved by the renal morphology. The scarring does not resemble a healed pyelonephritis in that the diseased foci appear to be entirely vascular in distribution and there is no post-inflammatory endarteritis.

FIG. 234.—The kidney surface is rough and granular, and the kidney bulk is reduced by about one-third. The ureter is dilated and its wall grossly thickened.

The characteristic features of these two cases were a dilated ureter with marked hypertrophy of the muscle coat, particularly in the lower third, a normal ureteric orifice on cystoscopy, and absence of resistance to the introduction of a full-sized catheter (*Fig.* 235).

DISCUSSION

Aetiology.—There is much confusion in the literature on the aetiology of megalo-ureter. The subjoined classification, suggested by Campbell (1938), although it does not include achalasia, is of value.

A. Mechanical Causes:—

1. Involving the Ureter :—

Congenital	*Acquired*
Stricture	Traumatic or inflammatory strictures
Reduplication	Stone
Blind endings	Tumour
Torsion, kink, cystic dilatation	Extra-ureteric pressure (stone, tumours, adhesions)
Diverticulum	
Valves, folds, vessels	
Abnormal insertion including ectopy	
Cyst	

2. Involving Bladder and Urethra :—

Congenital	*Acquired*
Vesical outlet obstruction (contracture, medium bar, medium lobe)	ditto
Diverticulum	ditto
Hypertrophy of trigone	ditto
Trigonal curtain	ditto
Prostatic and urethral cysts	ditto
Hypertrophy of veru	ditto
Posterior urethral valves	—
Stricture	ditto
Polypi	ditto
Phimosis	—
Extra-urethral masses	ditto
Abnormal openings (exstrophy, epispadias, hypospadias)	—
Penile torsion	ditto

B. Dynamic Causes (Neuromuscular Disease):—

Congenital	*Acquired*
Atonic cord bladder	ditto
Spasmodic cord bladder	ditto
Absence of muscular coat of the ureter	—
	Achalasia of the ureterovesical orifice

C. Inflammatory Causes (Long-standing Renal and Ureteric Infection).

The ureter is remarkable in that the longitudinal muscle layer is inside the circular. Numerous ganglion cells are found in the adventitia of the pars intramuralis and to a lesser extent in the lower third. The 'law of the intestine' holds good in the ureter, and if a wave of contraction meets resistance a hold-up takes place. The calibre widens to accommodate the hold-up and the walls hypertrophy in trying to expel it. When the hold-up takes place at the intramural portion the dilatation and hypertrophy are naturally most marked in the lower part of the ureter. As the condition progresses the dilatation and tortuosity spread to the middle and upper ureter and eventually to the pelvis and kidney. As a rule, therefore, the hydronephrosis is not proportionately as great as the hydro-ureter, although in the first case described here a very large hydronephrosis existed, but it is likely that pregnancy and the severe infection contributed to this. Incidentally, it is interesting to note that Caulk's patient (1923) had two full-time pregnancies and one miscarriage without any urinary disturbance, but in that patient the megalo-ureter was on the left side. Another feature of interest is that megalo-ureter has been reported in association with cardiospasm and Hirschsprung's disease (Hepler, 1937). The obliquity of the intramural channel prevents regurgitation during the act of micturition and the widening of the channel facilitates the propulsion of urine as the peristaltic ureteric wave reaches the bladder. Failure of this reciprocal arrangement, i.e., contracture of the juxtavesical ureter and at the same time dilatation of the intramural ureter, creates a dynamic barrier. The term 'achalasia' was introduced by Cooper Perry (1914) to describe the absence of relaxation of the sphincter muscle, a condition typically seen in cardiospasm and in Hirschsprung's disease. It was applied by Hurst (1931) to this type of megalo-ureter. His view was not universally accepted owing to the poor development of the circular muscle fibres in the intramural ureter : in fact, there is no circular sphincter muscle present. There appears, however, to be some support for his hypothesis that a breakdown in the neuromuscular co-ordination is responsible. On cystoscopy the active behaviour of the ureteric orifice is readily seen. After an escape of urine the orifice is pulled up and its lips tightly shut.

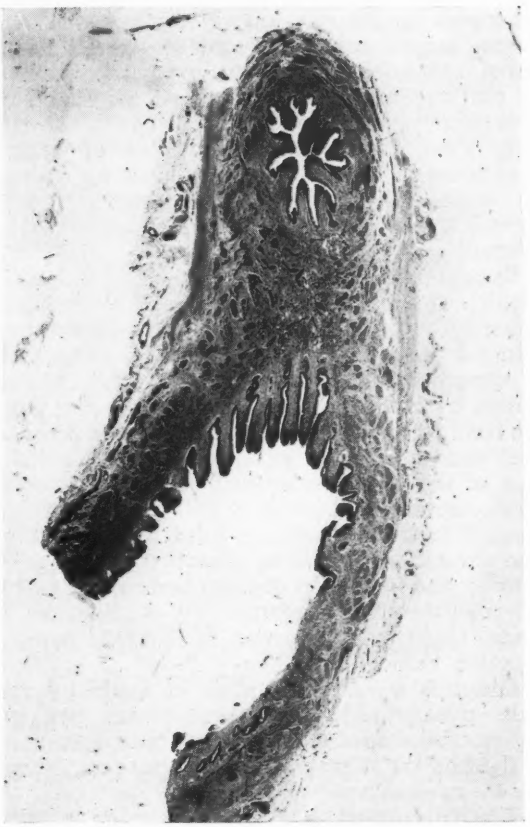

FIG. 235.—Section of the ureteric wall to show the hypertrophied muscle.

Learmonth (1931) has also demonstrated that stimulation of the presacral nerve causes a similar action, the contraction being often preceded by an efflux of urine. Hurst (1931) claimed that progressive degeneration of the ganglion cells of Auerbach's plexus is responsible for cardiospasm and suggests that a similar degeneration of the ganglion cells in the adventitia of the ureter may be responsible for this neuromuscular imbalance. In support of this view may be quoted the case of bilateral megalo-ureter reported by Kretschmer and Hibbs (1933) following post-diphtheritic paralysis, and also the case of megalo-ureter associated with spina bifida reported by Campbell (1938). Furthermore, there is the beneficial effect of presacral neurectomy in relieving the condition, an operation first suggested by Learmonth (1931), and the successful case reports by Ogier Ward (1933) and Berkman (1933).

Symptoms.—The symptoms are those of chronic urosepsis not responding to treatment. Pain may be present along the course of the ureter or in the kidney of the affected side. There were no symptoms in a case reported by Ockerblad (quoted by Hinman, 1935); and in the first patient here recorded no symptoms appeared until pregnancy developed, and in the second case a search for the cause of the hypertension revealed the existence of megalo-ureter. Campbell (1938) makes an interesting observation that one child in four suffering from chronic urosepsis has dilatation of one or both ureters; and in a series of post-mortems on children with megalo-ureter reported by Kretschmer and Hibbs (1933) in only 4 was the urinary pathology suspected before death.

Diagnosis.—The diagnosis is made by retrograde pyelography. The intravenous pyelogram may not be conclusive owing to poor secretion of the dye and the normal ureteric orifice on cystoscopy may be misleading.

Treatment.—The various methods employed for treating the condition are as follows: dilatation, meatotomy, plastic operation on the ureteric orifice, presacral neurectomy, and nephro-ureterectomy. Dilatation was employed by Berkman with a certain amount of relief of symptoms, but eventually neurectomy became necessary. Meatotomy was successful in Caulk's case, but it had to be performed at least three times and the ureter never returned to a normal size but still remained very dilated. Stretching of the orifice and sewing the mucous membrane of the ureter to that of the bladder was done by Thomson-Walker on Hurst's patient: the result was clinically very good, but no follow-up pyelogram was taken to estimate the ureteric condition. Learmonth (1933) suggested neurectomy and he advised dividing the nerves close to their entrance into the hypogastric ganglion, and by so doing only the lower end of the ureter was interfered with. Ogier Ward's case was very satisfactory, but the patient, a male, became sterile. There is no mention if Berkman's patient became sterile or not. Obviously nephro-ureterectomy is the operation of choice if the kidney is not worth saving. I can find no reference in the literature to reimplantation of the ureter into the bladder, an operation which appears to be worth doing if the kidney is healthy and if the patient is a male. By this operation also, extreme degrees of dilatation would probably subside more readily than after a presacral neurectomy.

SUMMARY

1. Two cases of megalo-ureter due to neuromuscular imbalance at the ureterovesical orifice are reported.

2. Hurst's theory of achalasia is discussed and fresh evidence submitted in its support.

My thanks are due to the Editor of the *Postgraduate Medical Journal* for allowing me to reproduce Figs. 233 and 234 and to Dr. S. B. Karani for supplying the notes of *Case* 2.

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A CASE OF TRAUMATIC PARARENAL PSEUDOHYDRONEPHROSIS

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THE following case report is presented because an unusual feature was found at operation, which would appear to make the report a useful addition to the literature on traumatic pararenal pseudohydronephrosis.

CASE REPORT

HISTORY.—The patient, H. B., a girl of 9 years, was admitted on Jan. 14, 1947. Having sustained a severe blow to the left side by falling down a deep hole, she was diagnosed as a rupture of the left kidney, on a basis of pain and tenderness in the left half of the abdomen and left loin, a very large retroperitoneal hæmatoma in the same region, and frank, gross hæmaturia. On conservative treatment she settled down and was discharged from hospital seventeen days later in good general health. Resolution of the hæmatoma had proceeded until it could be felt with certainty no longer.

The patient was readmitted on April 13, 1947. She had kept in good health since discharge, but her doctor had noticed that she had developed a scoliosis, concave

to the left. He had observed, in addition, a large smooth swelling on the left side of the abdomen.

On Examination.—The child was obviously in good general health. The existence of scoliosis, concave to the left, was confirmed. Inspection of the abdomen showed a swelling which appeared to involve the left half of the abdomen. On palpation, a smooth swelling was found, extending from under the costal margin to the interspinous line, and from the midline into the flank. The splenic flexure and descending colon could be made out as a band, tympanitic to percussion, crossing the mass.

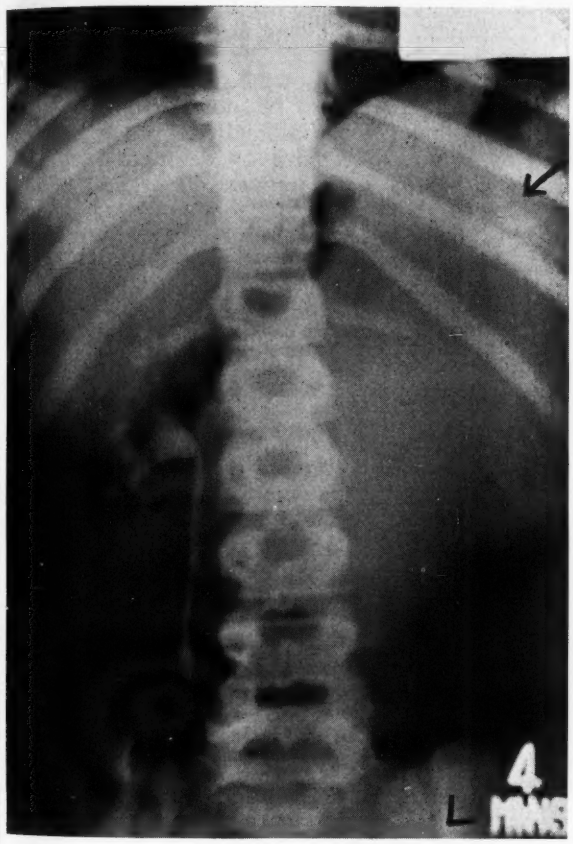

Fig. 236.—An intravenous pyelogram before operation. The sac of the pseudohydronephrosis is shown as the dense area to the left of the spine. The arrow indicates the dye outlining the renal pelvis, displaced upwards by the sac.

Intravenous Pyelography (*Fig.* 236).—The right renal pelvis was normal in outline and excretion time was normal.

Excretion time was normal in the left kidney. The pelvis was badly outlined and its direction could not be determined. It was sited high up under the left half of the diaphragm at the apex of an opaque area which represented the palpable mass. The renal mass extended from the level of the tenth dorsal to the fifth lumbar vertebra.

A tentative diagnosis of post-traumatic hydronephrosis was made and it was decided to operate on account of the size of the mass and because it was felt that a grave risk would be incurred if infection developed in such a large hydronephrosis.

At Operation.—The abdomen was opened by a left transverse incision extending from the ninth rib to just above the umbilicus, dividing all layers including peritoneum in the same line. This incision gave excellent exposure. A large cystic swelling (*Fig.* 237) was seen on the posterior abdominal wall, surmounted by the splenic flexure and descending colon. It was impossible to strip the colon medially with peritoneum as the wall of the sac and the posterior parietal peritoneum had become so closely knit together. Therefore, the colon was dissected off the sac leaving the anastomosis between mid-colic and left colic vessels intact, in close relation to the raised colon. Three pints of urine were aspirated from the sac. Careful dissection was commenced in an attempt to clear the sac and kidney intact, but it soon became clear that this would be impossible, with safety, particularly owing to the close relation between the sac and the aorta. The sac was opened anteriorly. Inspection of the inside of the sac showed that it was not a true hydronephrosis but a sac with a wall of fibrous tissue continuous with the lower pole of a normal-sized renal

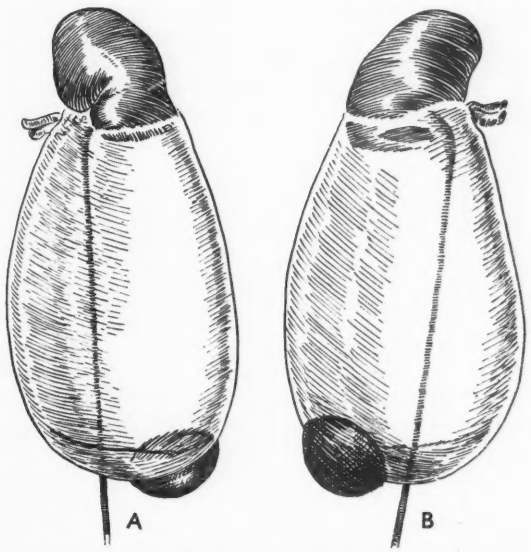

Fig. 237.—Semi-diagrammatic representation of the condition found at operation. A, Anterior view. The detached lower pole is seen in the wall of the sac, widely separated from the kidney. The vessel to this pole and the ureter are seen traversing the cavity of the sac. B, Posterior view. The open lower aspect of the renal pelvis is portrayed as being just visible through the sac wall.

pelvis. Through this sac the ureter ran without any attachments until it traversed the lower pole of the sac. The lower pole of the kidney had been severed from the main body of the kidney at the original injury and now lay at least three inches away from the main body, forming part of the lateral wall of the sac. The detached pole was still viable and a small vessel of supply could be seen running across the cavity of the sac to it. The kidney, ureter, avulsed lower pole, and anterolateral wall of the sac were removed in continuity, but the majority of the posteromedial wall had to be left in situ owing to its close relationship with aorta, pancreas, etc. The renal vessels, buried in the fibrous tissue of the sac wall, were dealt with by mass ligature. The site for the ligature was determined by guess in that, after clearing the kidney, the sole remaining attachment of fibrous tissue was judged to encompass the vessels.

During this procedure there was considerable oozing, but loss of blood did not appear to be excessive. However, the procedure was tedious and blood-loss was difficult to estimate.

A drain was inserted through a stab wound in the flank to allow escape of blood if further oozing did occur. The laparotomy wound was closed in layers.

Progress.—One pint of blood and two of saline were administered intravenously post-operatively. Thereafter the oral route sufficed for fluids.

Convalescence was uninterrupted and the little girl was allowed up four days after operation. Physiotherapy was instituted to correct the scoliosis. She was discharged on May 9, 1947, with sound healing of the operative wound. Follow up has shown that she has maintained excellent health up till October, 1947.

DISCUSSION

The unusual feature in this case, relative to other cases of traumatic pararenal pseudohydronephrosis, was the position of the ureter. The ureter traversed the cavity of the sac so that, from the renal pelvis to the lower pole of the sac, it lay free, surrounded only by urine. A study of the literature (Bonniot and Douillet, 1934; Crabtree, 1935; Woodruff and Rupert, 1939; Johnson and Smith, 1941; and Baretz, 1944) fails to produce any evidence of previous reference to this arrangement of the ureter in similar cases of pseudohydronephrosis.

It is not proposed to discuss the pathogenesis of traumatic pararenal pseudohydronephrosis as this has been summarized recently by Crawford Stanley (1947), and is dealt with at length by Crabtree (1935). It is interesting to consider how the ureter came to lie in the manner described in the case report. The majority of cases reported describe the ureter as buried in the fibrous tissue wall of the sac. Presumably this position is the result of the siting of the ureter in the periphery of the original hæmatoma. As the periphery of the hæmatoma organizes, it includes the ureter in the organizing portion. It is probable that this very inclusion causes the ureteric obstruction which forces urine through the unhealed rent in the kidney or pelvis. In the case described above it appears that the ureter must have lain centrally in the original hæmatoma and that only the lower end of the ureter was involved in organizing periphery of the hæmatoma. As the urine flowed from the unhealed rent in the lower pole of the renal pelvis, converting the central (and, presumably, still fluid) portion of the hæmatoma into a pseudohydronephrotic sac, the ureter contained in the sac would be rapidly surrounded with urine. This explanation would be compatible with the operative findings. The fact that the hæmatoma had resolved to such an extent before the first discharge from hospital that it could no longer be felt with certainty is not a strong argument against the above explanation, since it would not be necessary for the hæmatoma to be large for the condition to develop.

Certain points in the operative approach which have not been stressed in the literature may be discussed, as they appear to be of importance. It has surprised me greatly to find that, in most cases, the operative approach has been through a loin incision. In view of the size which the sac is liable to attain, this approach is extremely limited. Certainly, in the case now reported, the operation could not have been completed through such an incision. When it is remembered that the kidney was pushed high under the diaphragm, and that the sac wall had become one with the posterior parietal peritoneum, it is obvious that a transabdominal approach was necessary so that all dissecting could be done under direct vision. The unilateral transverse incision of the type described in the case report gave such a satisfactory exposure that it would appear to be the soundest to utilize in similar cases. It has the additional advantage of being unlikely to leave weakness of the abdominal wall.

During the operation on the case reported above an initial attempt was made to dissect out the sac intact. It would appear that this was a mistake as the sac itself is of little importance. As the wall was of fibrous tissue, it could have been of importance in itself only if it had had a lining of epithelium. Crabtree (1935) points out that in his own three cases and in the only two of the cases he collected from the literature in which special mention was made of this point, there was no evidence of a lining of epithelium. In the case now reported this point was not checked specifically, but no harm came from the fact that about half of the sac wall had to be left in situ. In general, lack of success from operations on fully developed pseudohydronephrosis has been due to failure to remove or defunction all renal tissue, either by actual removal or arterial ligation. In view of these facts, the following suggestion is made as to the wisest procedure after displaying the sac. After aspiration of the sac, it should be opened widely in order to allow a rapid determination of the site of the kidney and any detached fragments thereof. If nephrectomy appears possible without too much trauma, this should be performed, removing only such portions of the sac as render the operation simpler. The rest of the sac, save for such portions as can be removed easily piecemeal, should be left in situ and drained through a separate stab incision to avoid further hæmatoma formation. This drainage would be all the more important if infection had been present. If a gross degree of trauma could be expected from an attempt at nephrectomy, the kidney should be defunctioned by mass ligature of its vessels, which would be buried in fibrous tissue. In either case, of course, a careful search for detached renal fragments would require to be made and these fragments removed or defunctioned.

SUMMARY

1. A case of traumatic pararenal pseudohydronephrosis is described, in which the ureter traversed the cavity of the sac.

2. A suggestion is made as to the manner in which this siting of the ureter occurred.

3. Certain points of operative detail are discussed.

My thanks are due to Professor C. F. W. Illingworth for advice in preparing this report; to Mr. G. Donald, the artist, who prepared the drawings; and to Colonel P. J. Hanafin, Medical Superintendent.

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FISTULA BETWEEN THE RECTUM AND URINARY TRACT

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Fistula between the rectum and urinary tract may arise from one of four causes—injury, inflammation, new growth, or congenital abnormality. Each type of lesion calls for special consideration.

1. FISTULÆ DUE TO INJURY

Excluding those due to obstetrical injury, which are not here discussed, incidence in different surgical centres seems to vary according to the number of perineal resections of the prostate performed. For example, at St. Mark's Hospital only 7 cases were treated in ten years (Norbury, 1938). In America the incidence is higher. This will be referred to again later.

Apart from this association, the only other circumstances in which one is likely to meet many cases of traumatic fistula are due to war on a major scale.

The problems arising in various types of lesion will be dealt with by describing and commenting on three representative cases.

a. **Fistula between Rectum and Ureter.**—This condition is rare. I have been unable to find a case recorded of traumatic origin, although a thorough search through the vast literature was quite impossible. Naunton Morgan (1938) described a case due to diverticulitis of the colon. An instance of recto-ureteric fistula is presented here :—

C. R. B., aged 24, was wounded on July 8, 1945. Amongst other wounds he had one due to a shell fragment which penetrated the left buttock near the coccyx and formed an exit wound in the left iliac fossa. He was treated at the 2/2 Australian Casualty Clearing Station, where at operation a " through-and-through " wound of the rectum and bladder was found together with two holes in the small bowel. Left iliac colostomy, suprapubic cystostomy, bowel suture, the insertion of a tube into the rectum through the anus, and the drainage of the coccygeal wound were performed. At about this stage he came under my care. A month later without any change in treatment his posterior wound had healed, some urine was coming per urethram and the colostomy was acting more efficiently. Urine was still obviously entering the rectum. A week later he had an attack of acute left pyelitis. The posterior wound again discharged urine. A cystogram showed no fistula between the bladder and rectum.

On Oct. 21 a urethral catheter was inserted and the suprapubic tube removed. Negative pressure drainage was maintained and a week later the catheter was removed. Normal micturition was promptly restored and the suprapubic wound healed rapidly. Despite this he had an unexplained evening pyrexia. There were a few pus cells in the urine which grew *B. coli communis* on culture. By Nov. 28 he was well, apyrexial, and ambulatory. All wounds were soundly healed. On Dec. 11 proctoscopy revealed fluid in the rectum. This was shown to be urine. Indigo-carmine injected into the bladder failed to appear in the rectum and cystoscopy showed a deformity near the position of the left ureteric orifice but no opening into the rectum. Indigo-carmine injected intravenously appeared in the rectum and the opaque medium entered the rectum during intravenous pyelography which also showed a dilated and tortuous ureter and a dilated renal pelvis (*Fig.* 238). The diagnosis of left ureterorectal fistula was then obvious. The vesicorectal fistula had closed, leaving a stenosis of the lower end of the ureter. Transplantation of the ureter into another part of the bladder was contemplated but the presence of a left inguinal colostomy and a suprapubic scarred area, recently healed, decided us against this. At this stage his coccygeal wound again discharged urine.

On Jan. 25, 1946, left nephrectomy was performed. A grossly dilated ureter with thickened walls was found and a large pyonephrosis with pyelonephritis existed.

Fig. 238.—Intravenous pyelogram in a case of left recto-ureteric fistula.

Post-operative convalescence was uneventful. It took some six weeks for the coccygeal wound to heal completely. The colostomy has since been crushed and closed. He now leads an active life.

Several lessons were learned from this case. First, diagnosis was difficult in the presence of the rectovesical injury. Drainage of the bowel and bladder and a policy of non-intervention were followed by healing of the bladder wound. The ureteric injury persisted because of the stenosis distal to the opening. Transplantation of the ureter would have been possible under better circumstances. In this case ascending renal infection made the outlook for that kidney hopeless. Prompt nephrectomy not only resulted in healing of the rectal wounds but also in the prevention of serious systemic disturbance arising from the kidney condition.

b. **Rectovesical Fistula.**—While not rare, individual surgeons do not see this condition frequently. Ewell and Jackson (1941) state that in traumatic cases, half are the results of operations and that

one-third heal spontaneously. Wilhelm (1944) found that out of 6 cases due to operative mishaps, 3 healed by relatively conservative measures. So commonly do fistulæ occur after perineal prostatectomy—always, it is claimed, due to inexperience on the part of the surgeon—that several well-recognized methods of treating them have been developed. These fistulæ are inconstantly called rectovesical and recto-urethral.

Suprapubic cystostomy with constant suction drainage is essential in most cases. Secondly, the passage of fæces through the rectum must be controlled. This may be achieved by diet and drugs which aim at securing constipation. At the same time the sphincter ani is dilated. Many prefer an immediate colostomy. Some fistulæ heal by these relatively conservative and simple procedures. Suprapubic drainage of the bladder and dilatation of the anus resulted in spontaneous closure in many fistulæ due to war wounds, according to the records of the Medical Department of the United States Army in the first World War (1927). Direct operative measures are sometimes necessary, however.

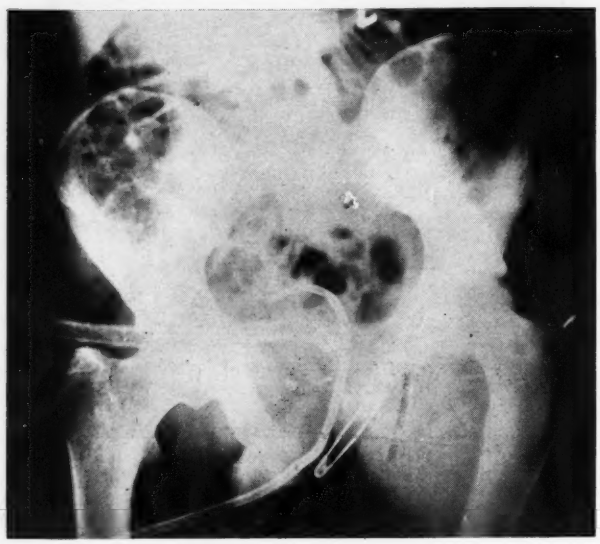

FIG. 239.—Shell wound of pelvis with gross bone and visceral damage and rectovesical fistula.

In America the method used by Young and Stone in 1913 (quoted by Davis, 1921) is recommended by many surgeons in that country. By an approach through the perineum the rectum is separated from the urethra or bladder. The urethra is sutured over a sound. The anal sphincter is then dilated and the rectal mucosa distal to and including the opening of the fistula is removed. The cut end is then sewn to the anal margin without tension. Thomson-Walker (1936, p. 564), on the other hand, writes: "Radical treatment consists in attempting to close the fistula by operation. This has been performed through the rectum after dilatation of the anal sphincter, with some successes, and a few cases have been operated upon through a suprapubic cystotomy wound. These methods are, however, inferior to the perineal route practised by Zuckerkandl." In the latter method both viscera are repaired and kept apart with gauze packing.

Fistulæ due to war wounds are different to those arising from operation in that the area is infected by the time the wounded man arrives at the first surgical centre and the wounds are more extensive. Usually the fistula forms but one of a number of serious wounds requiring even more urgent attention. Immediate repair is therefore out of the question. Nevertheless most rectovesical fistulæ of traumatic origin heal spontaneously. F. Legueu (1917), in a consideration of 43 cases of bladder injury due to war wounds, found that 15 rectovesical fistulæ existed. Of these, 12 healed spontaneously in four months or less.

J. R. C., aged 26, was wounded in June, 1945, receiving a penetrating lesion of the pelvis, the shell fragment entering the right groin and leaving the body in the region of the left buttock. The anterior wall of the bladder was blown away, the symphysis pubis shattered (*Fig.* 239),

FIG. 240.—Same case as in *Fig.* 239 after bladder reconstruction.

there was a perforating wound of the rectum and abdominal cavity, and two holes were demonstrated in the small bowel. Skilled treatment on the part of the forward surgeons dealt with the wounds—the bowel was sutured, left iliac colostomy performed, the bladder partially reconstituted around a catheter, and the cave of Retzius packed to control bleeding. Soon afterwards Lt.-Col. Turnbull, at the 2/11 Australian General Hospital, reported that there was a hole, 1½ in. across, between the rectum and bladder, that urine poured out of the wound and that a catheter introduced through the penis entered the rectum.

On arrival at 113 C.M.H. at the beginning of August he was desperately ill. He lay in a pool of urine and had profound toxæmia from an abscess of the groin. At operation Major Brooke Moore found that the anterior half of the bladder was missing, the cavity being half full of necrotic fragments of pubis, constituting what Legueu calls "une fistule ostéopathique". Most of these fragments were removed and the hole into the rectum could then be seen. Several stitches were inserted to draw

the margins together. The lateral walls of the bladder were mobilized and brought around to the front so that they could constitute a new anterior wall. They were sewn around a large tube. Constant suction was applied to this. This worked efficiently and in two weeks gradual improvement could be seen. Then two months later more bone was removed from the bladder, the anterior wall of which had not held together completely. This was sutured again. A month later he was able to get out of bed. Since then the history has been one of gradual improvement, with relapses due to malaria and vesical calculi. The urethra was almost completely stenosed in its prostatic part but has been dilated to normal dimensions. The opening between rectum and bladder gradually became very small and invisible through a cystoscope. Urine still entered the rectum when the suprapubic tube was blocked. The bladder capacity was good (cf. *Fig.* 240) but constant suction inevitably caused contraction. The fistula has been repaired, but three attempts were necessary. The fistula was in reality one in which the rectum communicated with a cavity where the prostate had been, the latter having been blown out through the anus. Both the bladder and prostatic urethra had therefore to be repaired. The unsuccessful efforts taught us lessons. On both occasions the fistulous opening was closed, but insufficient tissue was placed between the rectum and urinary tract. The collapse of the symphysis pubis constricted the operative field and reduced the amount of tissue available. Two weeks after the first operation an enthusiastic orderly washed out the colon distal to the colostomy, the anal sphincter being contracted. The thin partition between rectum and bladder gave way. After the second operation this partition was penetrated by the passage of a urethral catheter. At the third operation the rectum and urinary tract were isolated and separated as on the first two occasions. The fistula was excised and the two openings closed. The rectum was then mobilized, and the anterior wall pulled down, so that the repaired opening was removed as far as possible from the bladder. On the first occasion the open perineum was packed. On the other two occasions the perineum was closed without drainage after the insufflation of penicillin and sulphanilamide powder.

It is obvious from this case that the bladder has great powers of healing. It is also clear that it may be wise to wait patiently to allow the maximal amount of natural healing of such fistulæ. This applies at least to war injuries. The operative difficulties may be very great, but the widest possible separation of the holes in the bladder and rectum is essential.

c. **Recto-urethral Fistulæ.**—Much that has been said about the treatment of rectovesical fistula applies to this lesion. Cure by operation is easier because of the accessibility of the part, and in war wounds, as stated by Professor René le Fur (1919), is nearly always necessary. It will suffice then to record one case of traumatic fistula which has several features of interest.

P. T., aged 22, was seen at the end of January this year. Two months previously he had fallen from a truck and sustained a fracture of the pelvis. A suprapubic cystostomy had been performed because of the passage of urine per rectum. Subsequently air bubbled out of the suprapubic tube. When first I saw him he was passing very little urine per rectum, but had a reddened, tender bulging in the perineum and in the left adductor region. Local cellulitis was producing a profound toxæmia. A left iliac colostomy was performed and the two swollen areas incised. Infected urine came from each. An opening half an inch in diameter was found in the membranous urethra communicating with a similar aperture in the rectum, with the collection of fluid in the perineum and with the urine in the thigh. Extravasations of urine into the thigh are uncommon but the fracture involved the left ischiopubic ramus and was accompanied by a rupture of Colles's fascia. Through the opening in the urethra a catheter was inserted into the bladder. So marked was the toxæmia that efficient drainage was of paramount importance. Two weeks later the patient was taken off the Seriously Ill list and a week later still a catheter passed per urethram into the bladder without difficulty. This manœuvre stirred up an acute inflammatory reaction so that the catheter had to be removed. Urine again appeared in the rectum. The perineum was explored a second time. The rectum and urethra were separated more widely, the two holes sewn, and the intervening area packed with gauze soaked

FIG. 241.—Urethral stricture following cure of recto-urethral fistula.

in monacrine emulsion. After necessary adjustment of the suprapubic drainage several acute exacerbations of local inflammation occurred and urine appeared once only in the rectum. The condition gradually improved but every attempt to leave a catheter in the urethra was followed by acute urethritis. Thus stricture formation occurred (*Fig.* 241), but this has now responded to gradual dilatation at the hands of Lt.-Col. Earlam, who was able to leave a catheter in the bladder per urethram. There is no evidence of recurrence of the fistula and the final stage was the closure of the suprapubic wound and the colostomy.

The lessons are these: drain the perineum early to prevent infection; assist this by suprapubic drainage of the bladder and constant suction; in a clean field separate the rectum from the urethra and repair the wall of each; allow the perineum to fill in with granulation tissue or, if free enough from infection, reconstitute the muscular pelvic diaphragm.

2. FISTULÆ OF INFLAMMATORY OR NEOPLASTIC ORIGIN

Where the cause is inflammatory or neoplastic either the bladder or the urethra may be involved. Cunningham (1915) wrote that where the original lesion is in the rectum, growths, tuberculosis, diverticulitis, actinomycosis, and syphilis are possible causes. Where the urinary tract is the seat of the causative lesion, growths, tuberculosis, and trauma are the commonest causes. Many cases ascribed to diverticulitis are probably associated with lesions of the rectosigmoidal junction or last loop of the sigmoid colon. Thomson-Walker (1936) records an instance of a fistula being due to inflammation in a diverticulum of the bladder. The first stage of an inflammatory fistula is the formation of an abscess which finally ruptures into the neighbouring organ. Willan (1933) reported two cases where urinary calculi had ruptured into the rectum. Carcinomas of the rectum may invade the prostate and bladder but they rarely cause a fistula. Such tumours usually remain solid. Last century many instances of fistula between the bladder and colon were ascribed to neoplastic conditions when the real underlying cause, almost certainly, was diverticulitis. In Thomson-Walker's cases the latter condition accounted for ten out of thirteen fistulæ. Few of these are actually connected to the rectum, rather to the sigmoid colon. Lockhart-Mummery (1934, p. 203) writes that rectovesical fistulæ " are very uncommon, and when present are generally the result of operation ".

The development of these fistulæ is insidious. The first indication of even the causative lesion may be pneumaturia and the passage of bowel contents per urethram. An exacerbation of the cystitis, if the initial lesion be vesical, or the onset of acute inflammation of the bladder, if the starting point be the rectum, is all too prone to follow. Yet the rate of onset is variable and surprisingly little damage may result to the bladder for some time. Occasionally cystitis may precede the formation of the fistula when the bladder wall is being involved in neighbouring inflammatory changes. The signs and symptoms of the initial lesion are present whether obstructive and cachectic when due to carcinoma, or toxæmic when due to inflammation. Pneumaturia is finally one of the most constant symptoms, the gas bubbling out at the end of micturition. The passage of bowel contents per urethram varies in constancy and amount. In the centrifugal deposit undigested muscle and starch can be seen. Frequently the contamination is obvious macroscopically. The fistula is difficult to see either by cystoscopy or proctoscopy. Urine in the fæces can be detected by the hypobromite method.

If the pathological condition be cancer, no treatment other than palliative is likely to be of any use, but operative removal can sometimes be attempted. In cases due to causes other than cancer, preliminary suprapubic cystostomy and colostomy are indicated. The fistula may close spontaneously even without these measures if lavage of bladder and bowel be carried out. If not it must be dissected out or permanent colostomy maintained. The surgical approach for excision of the tract will vary with the position of the fistula.

It is not proposed to deal with fistulæ between the rectum and urethra of inflammatory origin, which are described in detail in standard text-books.

3. CONGENITAL FISTULÆ

I have depicted in this section, for the first time, all the different types of congenital fistulæ between the rectum and urinary tract to be found in the literature.

This type of fistula is rare, and is usually a complication of imperforate anus. Harrison Cripps (1882) estimated that imperforate anus occurred once in every five to six thousand births, and of these about one-tenth appeared to be accompanied by urinary fistula. Curling (1876) found the incidence 20 per cent, but Lockhart-Mummery (1934) states that all cases of imperforate rectum which he examined had urinary fistulæ. The various modifications of atresia ani are called atresia ani urethralis, atresia ani vesicalis, or atresia ani vestibularis, depending on the part of the urogenital tract involved. The minute openings are not easy to find (*Figs.* 245–247).

In some animals there is normally a communication between the rectum and urinary tract. In the Amphibia, gut, bladder and genital passages open into the cloaca. The same arrangement is found in Reptilia, but here there is commencing separation of the cloaca into urogenital and rectal portions. The vasa differentia which receive the ureters open into the anterior and not the posterior part of the cloaca. In Monotremes the genital ducts and ureter enter the anteriorly situated urogenital sinus which is now separate from the rectum and opens independently into the cloaca. On the ventral wall of the latter the penis or clitoris develops. Certain types of fistulæ in man have been likened to conditions found in other animals. Such speculations, while interesting, are frequently without meaning.

In a 5-mm. human embryo allantois and hind gut meet at an acute angle which contains the mesoderm of the urorectal septum. This septum grows between rectum and urogenital sinus, finally reaching the cloacal membrane. Wood Jones (1904) suggests that such a phenomenon is due, not to growth of any septum, but to the actual increase in size of the two cavities concerned. The passage between urogenital sinus and hind gut in a 5-mm. embryo cannot be more than 1 mm., so that the cloacal canal described in embryos of 8 mm. is probably only the unaltered original communication which is found to be obliterated by the 16-mm. stage, the cloacal membrane being then divided into anal and urogenital membranes.

The possible points of entry of recto-urinary fistulæ can be understood only by reference to the fate of the urogenital sinus. At one stage it receives the genital and excretory ducts, although the latter, probably by their own growth and expansion, actually contribute to the wall of the bladder in the region of the trigone. The urogenital sinus also receives the cloacal canal and is thus physiologically if not morphologically equivalent to cloaca. It ultimately forms the prostatic urethra and, to a small extent, the base of the bladder, and is prolonged forward under the phallic eminence (*Figs.* 242–244), forming

the definitive urethra which is completed, after rupture of the urogenital membrane, by the fusion of the genital folds.

The cloacal canal does not close if the ectodermal anal canal does not develop or if the caudal part of the rectum does not grow down to meet the latter structure. As would be expected, the most frequent anterior opening of fistulæ in the male is into the prostatic urethra (*Fig.* 245). Harrison Cripps wrote concerning the post-mortem finding in 10 male infants who had recto-urinary fistulæ. Six of the latter entered the prostatic urethra and 4 were connected to the bladder, 2 at the trigone, and 2 at the apex. This is a higher proportion of cases with bladder involvement than is usually reported. Fistulæ into the bladder are not mentioned by some authorities. Kermauner (1924) cites an instance of fistula into the bladder (*Fig.* 246), where failure of kidney development had occurred.

Fig. 242.

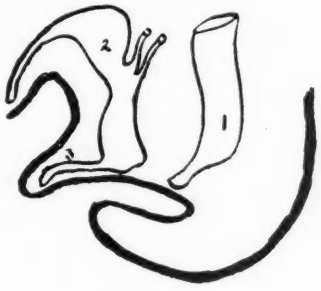

Fig. 243.

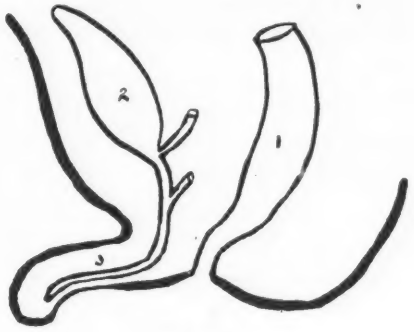

Fig. 244.

FIGS. 242–244.—Diagrammatic representation of separation of rectum from urogenital system.
Key to Figures 242–253. 1, Rectum; 2, Bladder; 3, Urethra; 4, Vagina; 5, Urogenital sinus; 6, Cloacal duct.

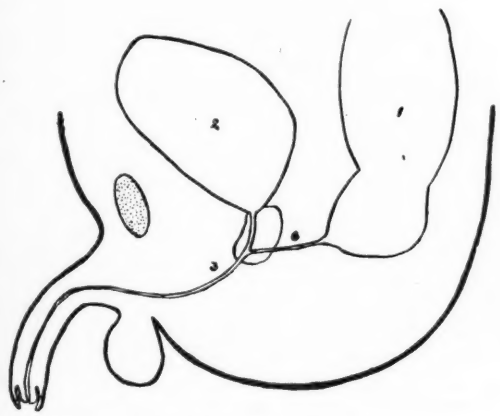

FIG. 245.—Atresia ani urethralis.

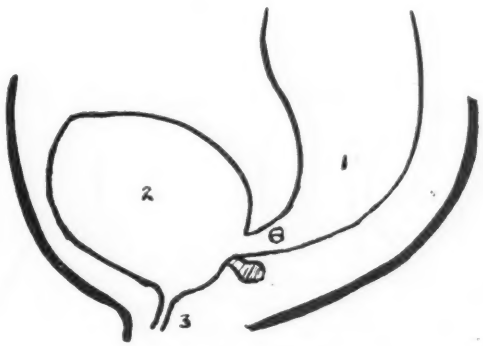

FIG. 246.—Atresia ani vesicalis.

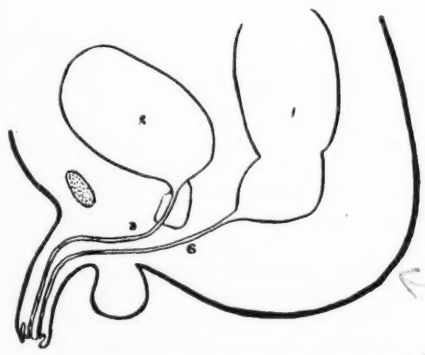

FIG. 247.—Atresia ani urethralis.

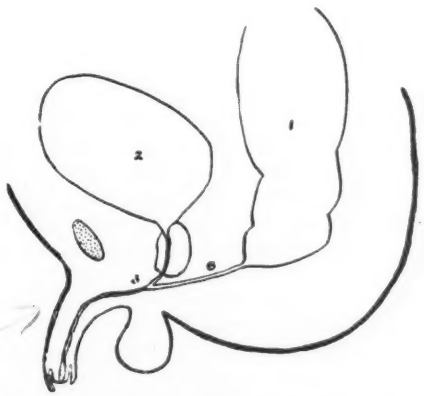

FIG. 248.—Double urethra and imperforate anus.

In a few rare instances the forward prolongation of the urogenital sinus under the phallic eminence is accompanied by a similar prolongation of the rectum, the anus being imperforate. The genital or urethral folds then enclose both prolongations so that the fistula may enter any part of the membranous (*Fig.* 247) or penile urethra, or, as in the case (*Fig.* 248) reported by Stieda (1903), the rectal prolongation may open on to the glans penis.

Fistulæ between the rectum and urinary tract are not so likely to occur in the female. However, occasionally the developing vagina becomes involved with the cloacal canal so that both bladder and rectum appear to empty into a distended vagina which is connected distally to the urethra. The hymen is imperforate. Examples of this are quoted by Kermauner (1924). In Ihl's case (*Fig.* 249) the contrary (1934, p. 61). Some cases (Harrison Cripps, 1882, p. 69) may survive for a year or more even though all the bowel contents may enter the urinary tract. Normal existence is all the more possible in the presence of recto-urinary fistula if the anus is patent. Thomson-Walker (1936) cites the case of a man who was still alive and well into adult life even though a recto-urethral fistula was

Fig. 249.

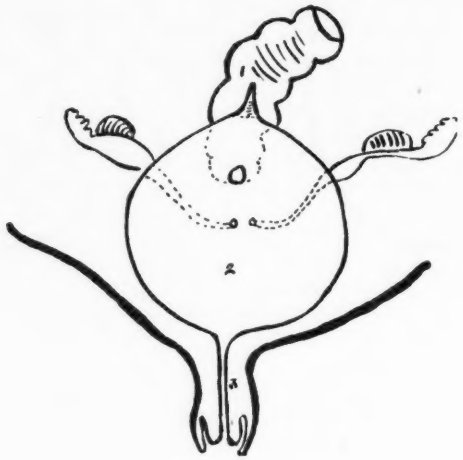

Fig. 250.

Fig. 251.

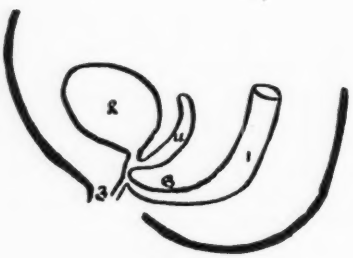

Fig. 252.

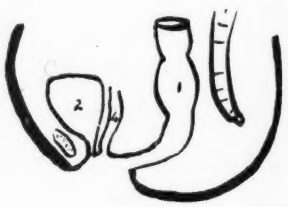

Fig. 253.

FIGS. 249–253.—Urinary fistulæ connecting with the rectum in the female.

above appearance was noted. In Grawitz's account the rectum and vagina were connected to the bladder (*Fig.* 250). Here the distal parts of the Müllerian ducts had not fused, so that each duct opened separately into the bladder. Vaginal development may occur with imperforate hymen, there being no connexion with the cloacal canal (*Fig.* 251). Keith (1908) depicts a case of recto-urethral fistula in a female, the vagina opening into the urethra more proximally (*Fig.* 252). In Mackenzie's case (1906) a more definite cloaca was present (*Fig.* 253).

Berenberg-Gossler (1913) emphasized the possible fistulous connexion between the bladder and other parts of the gut.

Clinical Features.—The fistula frequently goes unnoticed since the surgeon's attention is focused on the imperforate anus and rectum. Sometimes the urine is obviously stained dark with meconium. By no means all the bowel contents enter the urinary tract, the fistulous opening being small and often supplied with an imperfect sphincter (Fraser, 1922). Such a condition is not incompatible with life, despite the statement of Lockhart-Mummery to the present. Abbe (1908) writes: "Nature is sometimes tolerant of these defects and infection does not always ascend the ureters."

Treatment.—The attitude of surgeons to these abnormalities has become more optimistic with the years. Harrison Cripps (1882) discussed the justification for operating at all, saying that "the only effect of successful surgical interference is to condemn the infant to a life of suffering from a contracted anus or an artificial opening in the groin". Despite this he considered the operation justifiable.

Many fistulæ close once the obstruction to the rectum has been removed. A discussion of all the technical details in achieving this is outside the scope of this paper, but briefly, the main points are these: no blind puncture of the rectum should ever be done; open dissection should be successful since the end of the gut usually lies below the level of the peritoneal reflection; colostomy is avoided except as an urgent device to overcome the obstruction, all other measures having failed. If the fistula does not close spontaneously, it must be dissected out. This should form part of the operation at

which the rectum is mobilized and brought to the perineum. Anal stricture and incontinence are possible complications. The closing of such fistulæ is aided by the absence of chronic infection and scar tissue.

The immediate surgery of the traumatic fistulæ was performed by several surgeons. In their final cure I had the invaluable assistance of Lt.-Col. M. S. Earlam and Mr. A. Carrodus.

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ACCESSORY URETHRAL CANAL IN THE MALE

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LATE GRADED SURGEON, R.A.M.C.

THE male genitalia are subject to a number of congenital abnormalities; this paper is concerned with accessory urethral canals, of which there are several varieties. The term ' double urethra ' has been used to describe almost all of the anomalous channels found in the neighbourhood of the urethra and it is considered better to drop the term altogether, since its use adds to the confusion found on comparison of the existing literature. De Berne Lagarde (1932) and R. Meyer (1911) consider that only cases in which the accessory canal lies above the true urethra should be termed double urethra.

CLASSIFICATION

Various classifications of accessory urethral canals have been adopted, each based on structure, since there is little agreement as to the mode of their development. Classifications have been presented by Taruffi (1891), Oudard and Jean (1921), Young (1926), Chauvin (1927), and de Berne Lagarde (1932). That of Taruffi has not been bettered and is given here as quoted by Lowsley (1939) :—

Type 1.—A seminiferous canal. There are two meati on the glans, through one of which seminal fluid is ejected, the other serving for the passage of urine.

Type 2.—Blind-ending canals, either terminating on the surface of the penis or opening into the normal urethra but not extending to the bladder. (*a*) Any accessory canal that opens on the surface of the penis but does not communicate with the normal urethra or bladder—common in hypospadiacs, opening usually on the under surface near the meatus, although a few have been reported on the dorsum. (*b*) Canals opening into the normal urethra but ending blindly in periurethral tissue. Embryological defects not to be confused with normal para-urethral ducts opening into the urethra and leading into short *culs de sac.*

Type 3.—The uriniferous canal—an accessory channel communicating freely with the urethra at some point in its course and opening on the surface of the penis.

According to Young (1926), in cases with dorsally situated urethral canals, the normal urethra (lower canal) shows no abnormality as a rule. The accessory canal lies above and parallel and separated by the corpora cavernosa. The orifice may be above the external meatus, usually on the glans or coronal sulcus, or it may be on the shaft of the penis. No previous case has been found in the literature in which a patent dorsal channel opened within the normal external meatus as in that about to be described.

The lining membrane of these accessory channels has been found to be similar to that of the normal urethra in the few cases in which it has been examined, except for appearances brought about by inflammatory processes.

COLLECTED CASES

The most recent collection, by Lowsley (1939), is of cases belonging to Type 2 (a) of Taruffi—those with accessory canals opening on the surface of the penis but not communicating with the normal urethra or bladder. Forty-two cases were found, and since then the following cases have been described which belong to this group : a dorsal canal ending blindly at the symphysis pubis (Fong, 1946). Two cases with dorsal canals ending blindly, the first at

the root of the penis and the second ending in the shaft of the penis (O'Heeron and Webster, 1946). A case of Type 2 (*b*) has been described by Haslinger

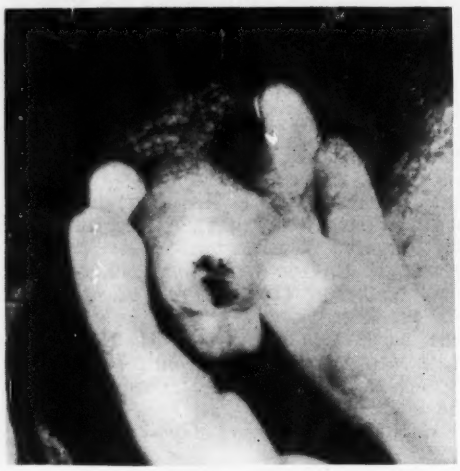

FIG. 254.—Photograph with the lips of the external meatus separated to show the junction of the normal urethra with the accessory canal.

(1939) in which an accessory canal was found to end blindly in the region of the symphysis pubis, and to open into the normal urethra within the meatus.

The next most complete review of the literature was by de Berne Lagarde (1932), who only found 38 cases of all types. Other collections have been

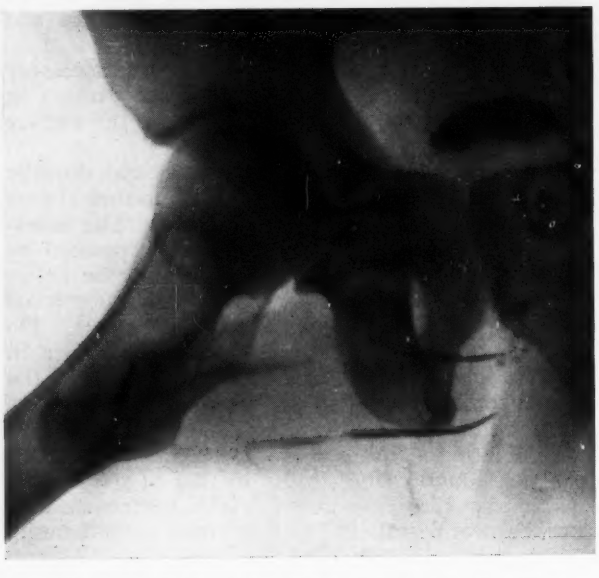

FIG. 255.—Radiograph after injection of radio-opaque substance. Interruption of the shadow is due to incomplete filling of the urethra. Lateral view.

made by Mackenzie (1916), v. Fault (1917), and Oudard and Jean (1921).

The case about to be described belongs to Type 3 of Taruffi, of which Mackenzie found 6 examples in the literature up to 1916, and 5 more have been described since. In Marion's case (1912) there was a supernumerary meatus in the glans above the normal orifice, which led to a channel that joined the normal urethra in the region of the bulb. Meisels (1893) had previously described a similar case in which the accessory canal met the urethra 8 cm. from the external meatus. Woods (1912), Poisson (1886), Dollinger (1890), and Djedurin (1905), described similar cases in which the normal urethra was joined by a long accessory canal, urine being passed through both channels. Another has been reported since by Bernadet (1923) in which a channel 10·2 cm. long ran from the bulbar region to a second orifice 3 mm. above the normal meatus, through both of which urine was passed. Haslinger (1939) has described a case in which an accessory canal opening on the dorsum of the penis was found to enter the bladder. Another has been found by Pytel (1934) in which a canal that opened on the dorsum of the penis entered the normal urethra in the region of the bulb. Two other cases have been described as complete double urethra in papers by Moreyra Bernan (1942) and Astani (1937); unfortunately it has not been possible to obtain copies of these papers and the cases cannot therefore be classified.

CASE HISTORY

Male, aged 41. Admitted to a British Military Hospital in the Middle East in December, 1946, under Captain A. W. B. Cunningham, late R.A.M.C., by whom the condition was discovered and investigated.

FIG. 256.—Radiograph after injection of radio-opaque substance. View from above, showing superimposition of channels.

He had gonorrhœa in 1933 and 1944 which was treated with irrigations on the first occasion, and by sulphonamide therapy on the second. Since then he had complained of intermittent pain in both loins, and of dysuria and frequency. There was no history of instrumentation.

External appearances were normal, and the patient himself was unaware of any anatomical abnormality.

The right testis and epididymis were tender and there was a urethral discharge. On parting the lips of the external meatus, which was normally situated and of normal size, a septum was seen which appeared to form the union of two urethral canals one above the other, as shown in *Fig.* 254. The dorsal channel appeared to be the source of most of the discharge; a probe passed into it easily and along the dorsum of the shaft of the penis where it could be readily palpated. A radio-opaque substance (Neo-iopax) was injected through the urethral orifice and was found to show two widely separated channels in the penis. The lower one was in the position and of the size and shape of the normal urethra. The upper one passed along the dorsum of the penis and then dipped between the crura below the symphysis pubis to join the normal urethra in the region of the bulb. From the junction the medium flowed up

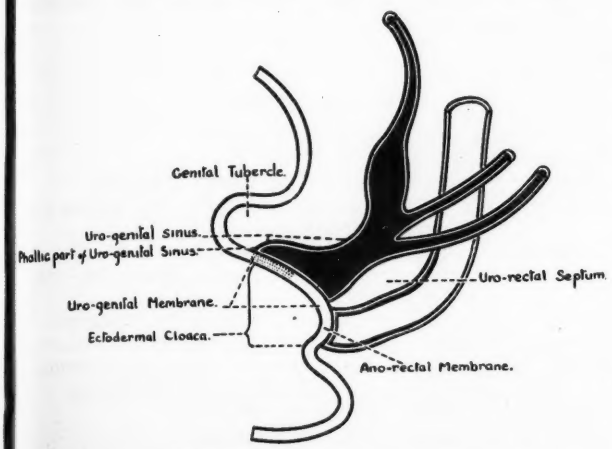

FIG. 257.—Diagram to show the differentiation of the urogenital membrane, and the phallic part of the urogenital sinus. Stippled area is that part which will proliferate to obliterate this portion of the sinus, or break down according to other authors. (*Modified from Keith*, 1933).

the prostatic urethra to the bladder. Seen from above, both channels were in the median plane and superimposed (*Figs.* 255, 256). Dye injected into the abnormal canal was voided through the normal urethra. Intravenous pyelography revealed no abnormality. A radiograph taken of the penis during the passage of this radio-opaque urine showed it to flow along both channels. Cystoscopy and retrograde pyelography (P.H.S.) showed no abnormality in the bladder, no accessory openings, and no abnormality of the upper part of the renal tract. Renal function was normal. A prostatic smear showed pus cells +, epithelial cells ++, a few diplococci, and many other organisms. There were no other congenital defects.

The patient was given 400,000 units of penicillin in three days and a course of 40 g. of sulphathiazole. This was followed by instillations of 2 per cent argyrol for one day, then of 5 per cent for six days. Finally he was given irrigations with 1–10,000 potassium permanganate twice daily anteriorly for two days and posteriorly for thirteen days, after which the discharge disappeared. There was a return of urethral discharge during an attack of bacillary dysentery in April, 1947, which disappeared when he recovered. No cause could be found to account for the pain in his loins, and this improved after treatment of his urethritis.

SYMPTOMS

The rare cases in which the accessory canal enters the bladder usually complain of partial incontinence, as the canal is not within the sphincter mechanism of the bladder. Most of the remaining types are asymptomatic unless they become infected. Of the 42 cases collected by Lowsley (1939), 26 had gonorrhœa, and the case described was brought to notice by the same complaint. The difficulty in treating these infections of accessory canals, especially those which end blindly, has been emphasized by O'Heeron and Webster (1946). The present case was no exception, but responded to prolonged treatment. The fact that so many of the known cases have been brought to light in this way suggests that anomalies of this kind are very much more common than a review of the literature would imply.

ANOMALIES IN THE FEMALE

Similar anomalies occur in the female urethra but are very rare. Five cases of accessory urethral channels have been found. Four are quoted by Stevens (1936) and another has been reported by Schinagel (1936). In only one of these did the accessory urethra enter the bladder. Epispadias in the female is more common than is generally supposed, 79 cases having been collected by Davis (1928).

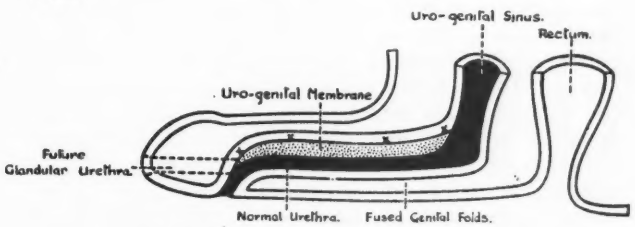

FIG. 258.—Diagram to show the development of the urethra prior to the formation of the glandular portion. The stippled area corresponds to that in *Fig.* 257. The line x x represents the obliterated phallic portion of the urogenital sinus, or the line of the accessory canal should it persist. If the urogenital membrane is considered to break down the stippled area would normally be absent.

EMBRYOLOGY

There is wide divergence of opinion as to the mode of development of the male urethra, which may be attributed to argument based on the method of comparative anatomy. There is no part of the body which shows such variation of development among the other members of the animal kingdom. In these circumstances it is only possible to follow the most universally accepted descriptions.

It is necessary in the first place to recognize the formation of an endodermal cloaca at the terminal part of the hind gut of the embryo; the extremity is turned round so that the allantois is carried towards the umbilicus, where it takes no part in the formation of the bladder. A depression corresponding to the position of the endodermal cloaca on the outer surface of the embryo is the ectodermal cloaca. The tissue between the two is the cloacal membrane. The endodermal cloaca becomes divided into two parts by the urorectal septum, formed probably from the ridges which give rise to the genital ducts. The anterior part of the endodermal cloaca is now composed of an upper part—the vesical primordium, and a lower part—the urogenital sinus. The cloacal membrane has been divided into an anterior urogenital membrane and a posterior

anorectal membrane. The urogenital sinus is then further divided into a pars pelvina and a pars phallica by depression of the middle part of the ventral wall of the cloaca (Felix, 1912).

"As the phallic portion of the urogenital sinus elongates in the sagittal plane it encroaches on, or is drawn into, the under surface of the genital tubercle [as shown in *Fig.* 257]. At the same time the endodermal lining of the urogenital membrane proliferates actively, especially in its anterior part, and the cavity of the phallic portion of the sinus is more or less obliterated. The proliferation in the part of the sinus related to the genital tubercle forms a solid urethral plate of epithelial cells. Shortly after the 16-mm. stage the posterior part of the urogenital membrane breaks down, and a communication, the primitive urogenital orifice, is established between the phallic portion of the urogenital sinus and the urogenital sulcus (ectodermal cloaca). The anterior part of the latter extends on the ventral aspect of the genital tubercle as the urethral groove on the surface of the urethral plate."

The above quotation is from Hamilton, Boyd, and Mossman (1945). Other descriptions state that the endodermal lining of the cloacal membrane breaks down, thus opening the pars phallica which becomes the floor of the urethral groove (Keith, 1933). There follows the formation and fusion of the genital folds which arise at the sides of the urethral groove, and unite over it from behind forwards to form the urethra as far as the coronary sulcus. A cord of epithelial cells then grows through the glans to reach the distal extremity of the urethra, and later becomes canalized to form the glandular portion of the urethra, which now comes to open at the tip of the penis (*Fig.* 258).

AETIOLOGY

The theories of development of accessory urethral canals have been summarized by Young (1926). A recapitulation of these would not be profitable.

Wood Jones (1904) has aptly remarked that "the growth of septa and partition walls which is so often invoked—and often wrongly invoked—as an explanation of various abnormal conditions, is founded on a misinterpretation of developmental changes, and, it would be urged, does not here occur, at any rate in the manner and to the extent that is usually described".

It is suggested that the dorsal accessory canals found in the male are due to a persistence of the pars phallica of the urogenital sinus from failure of the anterior part of the urogenital membrane to obliterate it by proliferation or failure to break down, according to the view taken of the fate of this structure. In the present case a single ectodermal ingrowth in the glans penis has united both with the normal and the accessory channels. Where two openings exist on the glans, separate ectodermal ingrowths have formed to meet each channel. The possibility of this mode of development seems first to have been considered by Grubenmann (1911).

SUMMARY

1. Accessory urethral canals in the male are classified, and cases not included in previous collections have been briefly reviewed. The condition is considered to be more common than is generally supposed.

2. A case is presented in which an accessory channel communicating freely with the urethra in the bulbar region opened with the normal urethra at a common external meatus.

3. A suggestion is made that dorsal accessory canals are due to the persistence of the pars phallica of the urogenital sinus.

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SUBPHRENIC ABSCESS

A CRITICAL SURVEY OF TWELVE CASES

By R. S. HUNT, Lt.-Col. R.A.M.C.

In the past sixteen months 12 cases of subphrenic abscess have come under my care. Ten of these were treated for their exciting cause, e.g., appendicitis or perforated peptic ulcer, at other hospitals, 3 followed operations that I myself had done, whilst 2 were in the final stages of recovery when they reached this hospital.

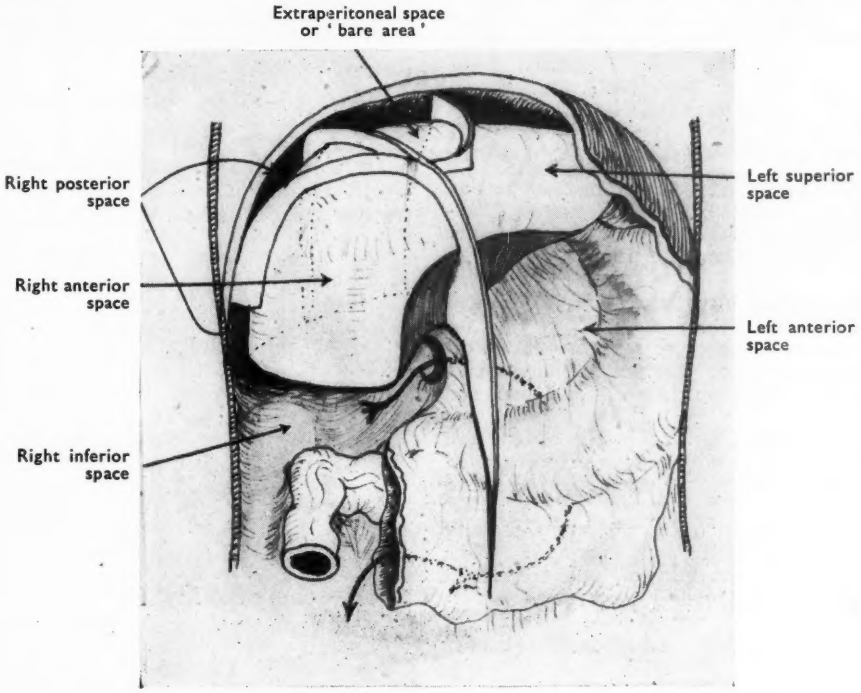

Fig. 259.—Diagram showing the anatomy of the region. Arrow passes through foramen of Winslow into left posterior space or 'lesser sac'.

The fact that all 12 have survived does not in the least minimize the gravity of the condition. Many of them have had the gravest complications, requiring a series of difficult operations to restore health and needing a long stay in hospital.

A critical survey of these cases may help in the prevention and treatment of subphrenic abscess and its serious complications.

Anatomy.—As a sound knowledge of anatomy is required when deciding on the best method of surgical approach, I submit the following short description and diagram (*Fig.* 259) which has always helped me to visualize the subphrenic spaces.

The subphrenic space lies below the diaphragm and above the transverse colon.

It is divided into upper and lower compartments by the liver and into right and left halves by the falciform ligament.

There are three spaces in the right half and three in the left. On the right side two spaces lie above the liver and are anterior and posterior. They are separated from each other by the right triangular ligament. The space below the liver on the right may be called the right inferior space (more commonly called the right kidney pouch).

On the left side one space lies above the liver and may be called the left superior space, whilst two lie below the liver and may be called left anterior and posterior spaces respectively. They are separated from each other by the lesser omentum, stomach, and anterior layer of the greater omentum. The left posterior space is more commonly called the lesser sac.

These six spaces are intraperitoneal, the seventh or extraperitoneal space corresponds with the 'bare area' of the liver and lies between the two layers of the coronary ligament. It extends over the upper surface of the liver and down the posterior surface just to the right of the midline.

CASE REPORTS

Case 1.—**Right anterior subphrenic abscess** (*Fig.* 260).

Private B., aged 22.

Feb. 5, 1947: A direct admission complaining of pain in the epigastrium and below the right costal margin.

History of "indigestion" for one year following an attack of jaundice, for which he was in hospital for three weeks.

Sudden severe attack of burning pain in the epigastrium two weeks ago which subsided in about twenty-four hours and was followed later by a gradually increasing throb in the same region.

On admission he had a large, smooth, hemispherical, tender swelling in the epigastrium. The liver was enlarged upwards and downwards. Breathing was painful and difficult and the temperature and pulse were raised. W.B.C. 30,000. Radiography: right diaphragm raised anteriorly; no pleural effusion.

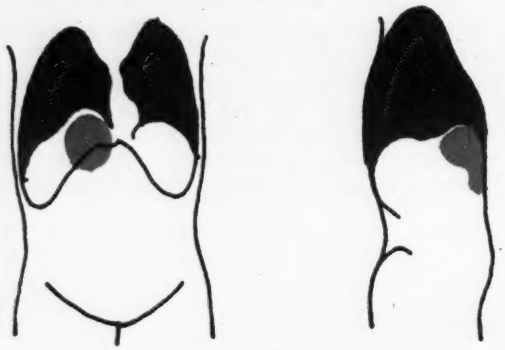

FIG. 260.—*Case* 1.

Drainage of abscess via high right paramedian incision by the extraperitoneal method. Abscess extended upwards between diaphragm and liver. Tube removed in 36 hours; 20,000 units of penicillin three-hourly up to 1,000,000.

March 5: One month after admission, discharged to Convalescent Depot.

Comment.—The exciting cause is uncertain. Possibly a perforated peptic ulcer; also possibly an amœbic liver abscess, for he had served abroad and had had dysentery.

The pus was yellowish-white and sterile on culture. The typical anchovy sauce is not always seen in cases of liver abscess. No other evidence of amœbiasis was found whilst he was in hospital.

No real criticism. Patient did not report sick early enough to assess the nature of the early symptoms. The condition was diagnosed on admission and treated at once on rational lines and he made a good recovery.

Case 2.—**Right posterior subphrenic abscess** (*Fig.* 261).

Quartermaster Sgt. H., aged 40.

Dec. 19, 1946: Peptic ulcer on the anterior wall of the first part of the duodenum perforated 25 hours before admission. Laparotomy and suture of the ulcer soon

FIG. 261.—*Case* 2.

after admission. There was much purulent fluid in the peritoneal cavity which was sucked as dry as possible.

A drain was inserted in the right kidney pouch and suprapubicly into the pelvis.

He was nursed in Fowler's position and given 20,000 units of penicillin three-hourly up to 1,000,000 units.

Jan. 2, 1947 (i.e., 14 days later): He showed a swinging temperature and pulse and complained of pain in the right shoulder, dyspnœa, and tenderness over the lower ribs at the back. X-ray examination showed a very high diaphragm on the right with some collapse of the lower lobe of the lung.

Jan. 9: Drainage of the abscess by resection of the 12th rib and an extraperitoneal approach. Pus culture revealed *B. proteus* and pneumococci which were penicillin-sensitive.

Comment.—Perforated peptic ulcer and appendicitis are the two conditions most commonly complicated by subphrenic abscess. Few people nowadays drain the peritoneum in cases of the former unless there has been considerable delay in operation or unless the pus is thick or contains food particles.

Some would argue that the flank drain in this case predisposed to the formation of the subsequent abscess, for except in a well-defined abscess a drainage tube will only drain the area in its immediate neighbourhood.

The week's delay between Jan. 2 and Jan. 9 was not justified.

Case 3.—**Right inferior subphrenic abscess** (*Fig.* 262).

Craftsman P., aged 20.

Admitted to a hospital in the Middle East on July 8, 1947. History of occasional attacks of pain in the R.I.F. for one month. Much increased for the past few days.

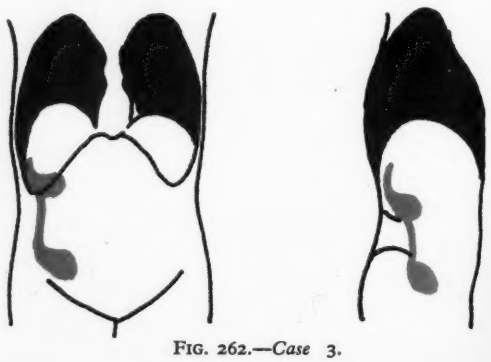

FIG. 262.—*Case* 3.

Diagnosed as an appendix abscess and possibly a pelvic abscess also. Treated by the Oschner-Sherren method.

July 10: Drainage of appendix abscess via a gridiron incision. A well-localized abscess was drained from which a free fæcolith was removed. He had a stormy convalescence.

Aug. 11: Evacuated to U.K.

Oct. 6: Admitted to this hospital after a month's leave for an interval appendicectomy. Free from symptoms and with no abnormal physical signs. Appendicectomy without drainage. This was a difficult case, for the appendix was a long retrocæcal one and there were still a great many adhesions.

He did not do well. Within a day or so he ran a swinging temperature and his pulse steadily rose. He complained of pain on breathing and was very tender in the right hypochondrium and in the right loin. A diagnostic aspiration was carried out, the needle being inserted in three directions, inwards and medially, inwards and upwards, and inwards and downwards. Foul-smelling pus was withdrawn from the medial direction. It was decided to drain the abscess by extending the original gridiron incision upwards and draining the abscess from its most dependent part. He was nursed in Fowler's position. This was on Oct. 17.

Oct. 23: It was clear that the drainage was imperfect. A right inferior abscess was then drained by resection of the 12th rib. He subsequently made an uninterrupted recovery.

Comment.—Diagnostic aspiration is a dangerous procedure. It should be possible to diagnose the presence of pus without it. It is easy to perforate some viscus or to spread infection.

An interval appendicectomy of such difficulty leaves a potentially infected bed and I think a small drain for 24 hours would have been advisable.

Fowler's position is difficult to maintain, especially in these days of staff shortage and with male nursing orderlies of the present standard. It is uncomfortable for the patient, and will not drain the inferior space unless the correct position *is* kept up. 12th-rib resection gives good access and better drainage.

Case 4.—**Right posterior subphrenic abscess** (*Fig.* 263).

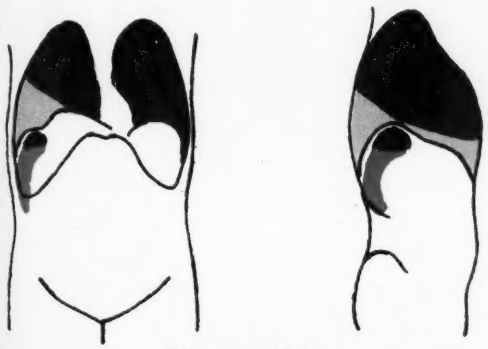

FIG. 263.—*Case* 4.

Quartermaster Sgt. H., aged 27.

Admitted to the Medical Division with fever, non-productive cough, pain in the right shoulder, and tenderness deep to the lower ribs on the right side. He was

FIG. 264.—*Case* 4. Lateral radiograph showing diaphragm very much raised. Diminished movement on screening.

treated with penicillin, sulpha drugs, and repeated aspirations of what was thought to be a lower lobe lung abscess with an overlying effusion. Culture showed the pus to contain *B. coli* and non-hæmolytic streptococci, the latter being penicillin-sensitive. He did not improve.

Similar history a year ago with admission to hospital with similar treatment. Some improvement but never complete recovery.

Feb. 22, 1947: Transferred to the Surgical Division. Temperature 102; pulse 120; respiration 26. W.B.C. 21,000. Radiography showed raised right diaphragm with diminished movement (*Fig.* 264), a small amount of gas beneath it, pleural effusion, and displacement of the heart to the left.

Feb. 28: Aspiration of chest fluid, sterile. Drainage of subphrenic abscess by resection of 10th rib in mid-axillary line.

Feb. 29: Drainage of small empyema via the same incision, the fluid having become infected with the same organisms as the abscess.

May 30: Radiography showed that right diaphragm remains slightly raised posteriorly and peripherally. There is slight resultant pleural thickening at the right lower zone.

June 3: Discharged.

Comment.—Cause not known. Barium meal showed no evidence of ulcer. Possibly a recurrent appendicitis, as this is the next most common predisposing cause.

I think it was a mistake to drain by resecting the 10th rib. It is unlikely to drain the most dependent part of the abscess this way and any pleural effusion runs a grave risk of being infected as indeed it did in this case. Better to resect the 12th rib and avoid this risk.

Case 5.—**Right posterior subphrenic abscess** (*Fig.* 265).

Captain B., aged 55.

July 19, 1947: Admitted to the Medical Division giving a history of "indigestion" lasting 1–2 weeks in attacks for the past 2–3 years. Free for several months until seven days ago, when another attack began. The day before, whilst on leave, had a very severe pain in the epigastrium which was eased by morphine and atropine and was diagnosed as acute cholecystitis.

When admitted to hospital the severe symptoms had subsided and treatment for cholecystitis was continued. He had a hectic temperature and pulse, with W.B.C. 21,000 and rigidity and tenderness in the right hypochondrium.

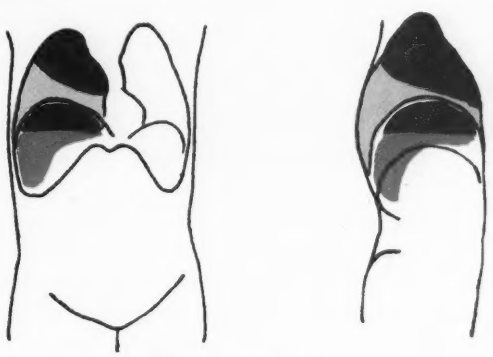

FIG. 265.—*Case* 5.

He did not respond to his treatment, but continued to run a fever, was toxic in appearance, and lost weight.

He developed signs of consolidation at the right base with a pleural effusion. W.B.C. rose to 30,000 and radiographs revealed an enormous subphrenic abscess with gas under the diaphragm (*Figs.* 266, 267).

He was transferred to the Surgical Division.

Aug. 7: Drainage of subphrenic abscess by resection of 10th rib posteriorly. A very large volume of foul-smelling pus was evacuated. Culture grew a coagulose-positive staphylococcus, penicillin-sensitive, and a non-hæmolytic streptococcus, also penicillin-sensitive.

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He improved considerably after operation but the pleural effusion (*Fig.* 268) which appeared to be loculated was aspirated daily and injected with penicillin. Within

FIG. 266.—*Case* 5. Anteroposterior view showing very large subphrenic abscess with gas.

a week his temperature and pulse rose again and culture revealed the effusion to be infected with the same organisms as the abscess.

Aug. 25: Empyema drained through the original incision, loculations being broken down with the finger. Drainage gradually diminished and the tube was removed on Sept. 26.

Oct. 2: Within a week of removing the tube the temperature and pulse rose again and radiographs revealed a further collection of fluid. This was drained by resection of the 8th rib in the mid-axillary line. He remained very ill for weeks and had become very introspective and very reluctant to have the tube removed "too soon".

Nov. 15: He complained of pain in the 10th-rib scar and the old abscess pointed and was drained again.

Dec. 20: Discharge had become insignificant and both tubes were removed.

Dec. 30: He was sent on a month's leave with instructions to return for final review of the gastric condition.

Comment.—Cause uncertain. Subphrenic abscess may follow almost any intra-abdominal infection. May have been a suppurative cholecystitis, but is more likely to have been a perforated ulcer. An early and accurate diagnosis might have prevented the condition.

Again I think it would have been better to drain the abscess by resecting the 12th rib rather than the 10th. The fact that it had to be drained a second time suggests that drainage was not adequate the first time.

In cases which show gas under the diaphragm a loculated pleural effusion is frequently seen, it is difficult to treat, and may require several drainage operations.

This patient's convalescence was interrupted by jaundice, which followed one of his many small blood transfusions.

It is always important to remember the exciting cause, hence the instructions to return for investigation and final assessment of the probable peptic ulceration.

FIG. 267.—*Case* 5. Lateral view showing very large subphrenic abscess with gas present.

It is also important to enforce the surgical treatment with correct adjuvant treatment such as diet, drugs, and breathing exercises.

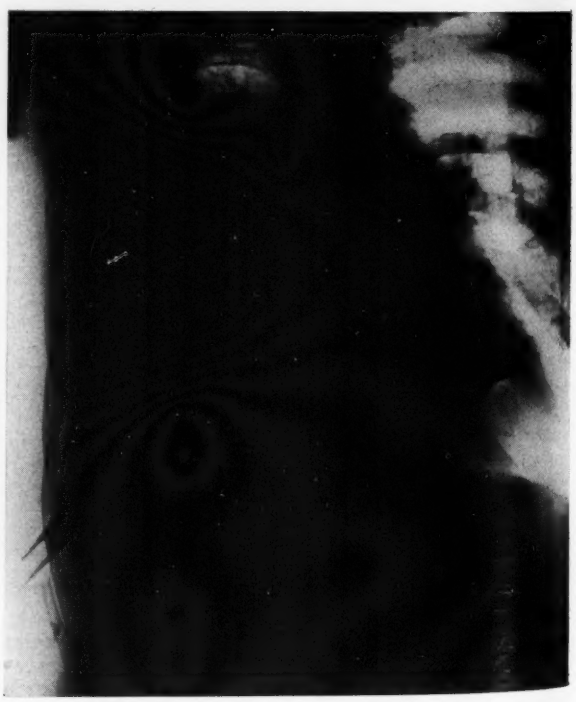


FIG. 268.—*Case* 5. Radiograph showing the subphrenic abscess drained. Pleural effusion has developed.

Case 6.—**Right anterior subphrenic abscess** (*Fig.* 269).

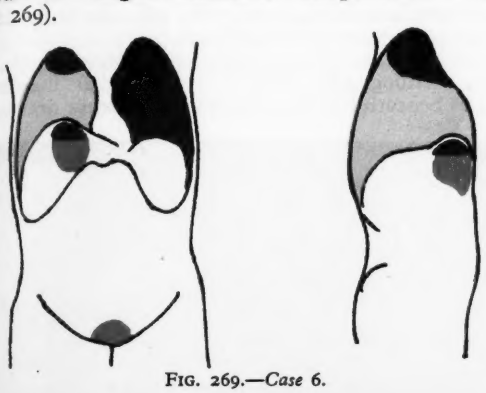


FIG. 269.—*Case* 6.

FIG. 270.—*Case* 6. Anteroposterior view showing raised diaphragm with gas present and massive pleural effusion.

Private T., aged 19.

June 17, 1947: Admitted with a perforation of an acute peptic ulcer on the anterior wall of the first part of the duodenum. He gave a history of three days' indigestion.

He was unusually shocked and of very poor physique. The perforation occurred four hours prior to his admission. Immediate laparotomy was performed and the ulcer sutured.

June 19: Embarrassed breathing, pain in the chest, and rising pulse and temperature. Signs of pleural effusion present. 10 oz. of sterile straw-coloured fluid were aspirated.

He also had pain in the abdomen, difficulty in micturition, and mucous diarrhœa. A tender fluctuating mass was felt P.R.

June 23: Pelvic abscess drained P.R.

June 30: Massive pleural effusion with loculations laterally; a raised right diaphragm with gas beneath it (*Figs.* 270, 271). Tender over the 10th rib in the midclavicular line.

Subphrenic abscess drained via a right subcostal incision.

Temporary improvement, but regression coincided with a gradual increase in the turbidity of the pleural fluid, which grew *B. coli* and penicillin-sensitive hæmolytic streptococci.

July 31: Drainage of empyema by resection of 9th rib posteriorly. Steady recovery.

Comment.—It is obvious, looking back, that the subphrenic abscess was present as long ago as June 19, and that had it been treated then the empyema might never have developed.

Here is another case where the pleural effusion was loculated and the subphrenic abscess contained gas.

FIG. 271.—*Case* 6. Lateral view showing raised diaphragm with gas present and massive pleural effusion.

Case 7.—**Left superior subphrenic abscess** (*Fig.* 272).

FIG. 272.—*Case* 7.

German P.o.W. J., aged 35.

History difficult to obtain, but said to have had indigestion for three days with sudden onset of acute pain in the epigastrium three hours before admission.

Oct. 25, 1946: Laparotomy and suture of perforated ulcer on the lesser curvature of the stomach.

Nov. 2: Eight days later, swinging temperature, embarrassed breathing, pain in the left side of the chest, and signs of pleurisy with effusion.

Nov. 6: Subphrenic abscess diagnosed. W.B.C. 20,000. Radiographs showed a raised left diaphragm with gas beneath it (*Figs.* 273, 274). Abscess drained by resection of the 12th rib posteriorly (*see Fig.* 276). Tube remained in for three days.

FIG. 273.—*Case* 7. Anteroposterior view showing gas in a subphrenic abscess on the left side with pleural effusion.

FIG. 274.—*Case* 7. Lateral view showing gas-containing subphrenic abscess on the left side.

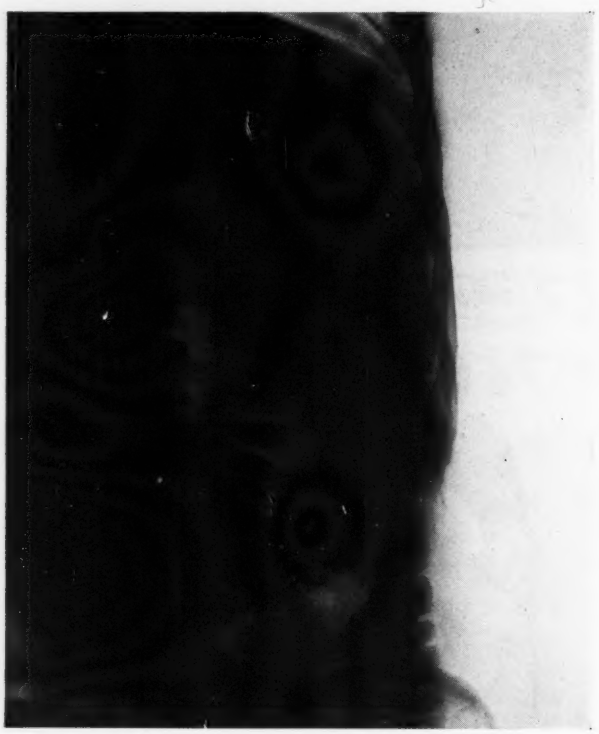


FIG. 275.—*Case* 7. Barium enema shows the presence of a fæcal fistula communicating with the abscess cavity.

Three days after removal of the tube he developed a fæcal fistula which refused to heal in spite of treatment.

Nov. 21: Transverse colostomy performed to exclude the fistula. Within a short time the spur sank and the colostomy no longer fulfilled its purpose.

The site of the fistula nor the reason for it could be understood. Barium enema revealed the fistula just below the splenic flexure, the barium being discharged into a large subphrenic abscess (*Fig.* 275). Further radiographs showed the fistula to be healing with stricture formation *distal* to the fistula. The abscess cavity was continually being contaminated by the passage of fæces.

Dec. 7: A further attempt to short-circuit the abscess and the fistula was made by anastomosing the transverse colon to the descending colon distal to the fistula. It failed in both its purposes.

Jan. 13, 1947: The abscess cavity walls had become fibrotic and a further attempt to promote healing was made by marsupializing the cavity by resection of portions of the 9th and 10th ribs.

The abscess cavity slowly healed but a narrow fæcal fistula persisted. This was almost certainly due to the stricture preventing the normal passage of fæces.

March 13: Closure of useless colostomy.

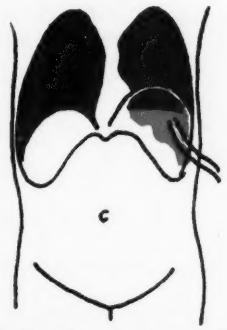
The fistula still persisted.

April 24: Division of the transverse colon distal to the side-to-side anastomosis (*see Fig.* 276). This was followed by complete healing and he was discharged to duty on June 1.

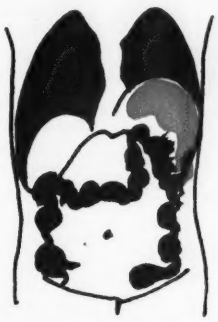
Comment.—This was probably a perigastric abscess sealed off from the general peritoneal cavity by the omentum and other adhesions and which subsequently spread upwards over the upper surface of the liver on the left side.

possibility was injury by the drainage tube pressing on the inflamed bowel.

It is interesting to compare this with the next case, where a very similar series of events resulted in even graver complications.



Drainage by resection of 12th rib. Tube in most dependent part of cavity.

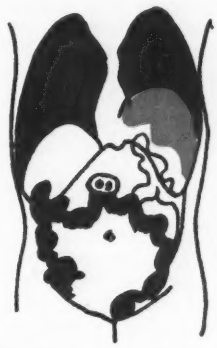


Fæces were discharged through drainage wound. Radiograph showed erosion of splenic flexure with fæcal fistula formation.

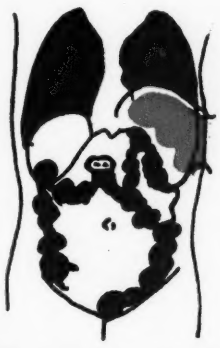
Transverse colostomy in attempt to by-pass abscess and prevent further fæcal contamination.

This worked well until the spur sank and—

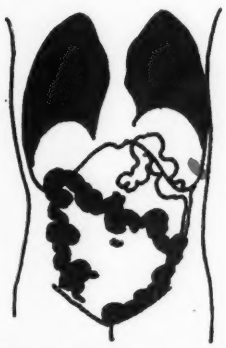
the previous state of affairs was re-established.



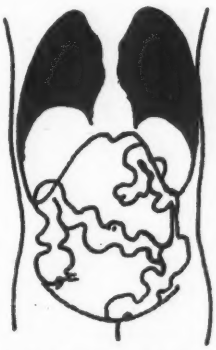
Transverse colon was anastomosed to the descending colon below the stricture.



This too failed. Abscess then marsupialized to get adequate drainage.



Healing then took place, leaving a narrow fæcal fistula only. Transverse colon then divided proximal to fistula.



Complete healing, with the mucous secretions of the blind end draining through the stricture into the distal gut.

FIG. 276.—*Case* 7.

The approach by resection of the 12th rib at the back was probably a mistake, and looking back I think it would have been better to attack the abscess by an extraperitoneal approach through a left subcostal incision.

It is a long way forward from the ribs at the back to an abscess above and in front of the liver, and to reach it one has to pass very close to the splenic flexure. This was realized at the time and the greatest care was taken to avoid injury to it. Operative trauma was, however, a possible explanation of the fistula formation. Another

In the early stages it was impossible to know that the fæcal fistula would heal and form a stricture by so doing, and the idea of a transverse colostomy was sound. It was unfortunate that the spur sank and thus prevented the colostomy from fulfilling its function (*see Case* 8 for comparison). If the condition of the patient would permit it, it would be far better to take a little extra time and do a true double-barrel colostomy.

It is remarkable that with infection below the diaphragm for so long and a pleural effusion present quite early on, no empyema developed.

Case 8.—**Left posterior and right inferior subphrenic abscesses** (*Fig.* 277).

Private L., aged 19.

Received as an air transfer from overseas in an extremely ill condition, wasted, toxic, and with very

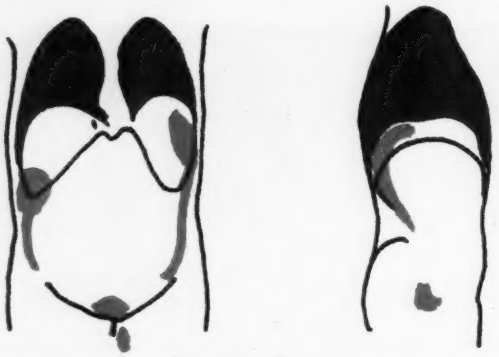


FIG. 277.—*Case* 8.

little will to live. He had: (1) A right paramedian incision discharging thin yellow pus from its lower end, and a transverse colostomy at its upper end. (2) An oblique incision across the left iliac fossa with a central sinus discharging the same yellowish pus. (3) Two parallel incisions in the left loin (as for a nephrectomy) both discharging pus and fæces. (4) A small gridiron incision in the right iliac fossa also discharging yellowish pus. (5) A healed incision in the left scrotum.

The history was as follows (*see Fig.* 279):—

March 19, 1947: First admitted to a hospital overseas complaining of throbbing pain in the epigastrium with marked flatulence and vomiting. Progressive deterioration with a swinging temperature and pulse. He developed acute tenderness in the left renal angle and an acute epididymo-orchitis on the left side.

March 23: Drainage of a left perinephric abscess. The notes go on to say that a quantity of foul-smelling urine was evacuated and that a carbuncle of the kidney was seen. The condition of the patient did not justify nephrectomy at that time.

March 25: Drainage of the scrotal abscess on the left side.

April 14: Left nephrectomy. Perinephric abscess had extended upwards under the diaphragm and downwards where it extended into the iliac fossa. This latter portion was drained by an oblique incision over the iliac fossa.

April 22: Developed fæcal fistulæ through both incisions in the left loin.

April 30: Drainage of a right paracolic abscess through a right paramedian incision. Transverse colostomy performed to exclude the fæcal fistulæ.

May 15: Developed a left femoral thrombosis.

At this stage he was flown home and was admitted to this hospital.

The exact state of affairs was obscure and his morale was such that it would have courted disaster to attempt any surgery at once. It was therefore decided to build him up as far as possible on adequate diet and careful nursing, and to let him get used to his new surroundings.

When the right time came I was anxious to clear up the sepsis before attempting any repair of the fæcal fistulæ, which the patient objected to far more than the sinuses discharging pus.

Investigations showed a right inferior subphrenic abscess to be present which was not draining adequately through the incision in the iliac fossa.

July 31: After a lot of persuasion this abscess was drained by resection of the 12th rib and subsequently cleared up (*Fig.* 278).

The pus exuding from the right paramedian incision was found to be coming from a large pelvic abscess, obviously draining uphill. It was decided to leave this and tackle the fæcal fistulæ first, however, for to do otherwise would have meant complete refusal on the part of the patient to consent to any further surgery.

Aug. 21: The fæcal fistulæ were explored through a left paramedian incision with a T-shaped extension out into the flank. Repair proved impossible and the colon was therefore excised from a few inches distal to the colostomy down to the first part of the sigmoid. The abscess under the diaphragm in the region of the splenic flexure and the fistulæ soon healed and with this result the patient's morale rose tremendously.

Sept. 4: The pelvic abscess was drained P.R. and cleared up.

Oct. 18: Sent on convalescent leave with a temporary colostomy belt.

Jan. 28, 1948: The blind end of the transverse colon was anastomosed to the blind end of the sigmoid via a transverse incision level with the umbilicus. A side-to-side anastomosis of the blind ends was performed. It was an extremely difficult procedure for there were many adhesions as the result of past sepsis and a considerable disparity in size between the transverse colon and sigmoid, the latter having been out of action for five months.

All that now remains is to close the colostomy.

Comment.—It is almost impossible to make any constructive comments on the early part of this long history. To have such widespread infection suggests a blood-borne origin. Possibly the infection spread from the left kidney to the perinephric space and thence both

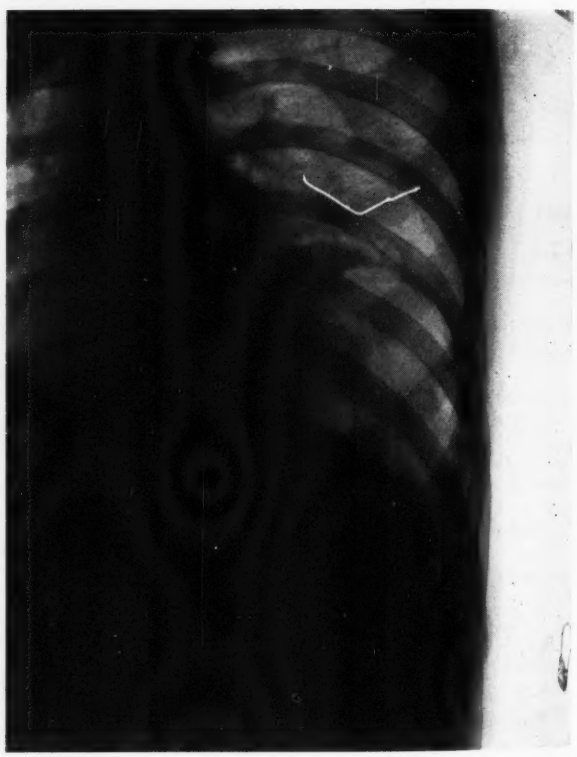


FIG. 278.—*Case* 8. Anteroposterior view showing the abscess drained.

upwards and downwards extraperitoneally as well as intraperitoneally.

This difficult case passed through the hands of several surgeons before arriving at a base in the U.K., which made it all the more difficult to assess and treat.

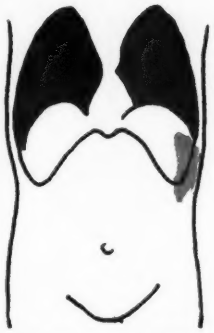
The liability to damage of the colon on the left side is again demonstrated, especially in the presence of sepsis.

The need for a true double-barrel colostomy as a method of excluding the colon is again shown.

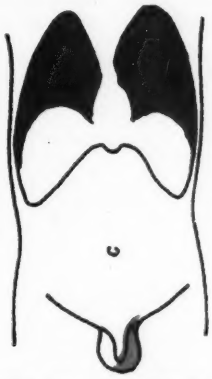
Two weeks ago pain in the right shoulder, which spread to the whole of his chest.

Two days ago had a small hæmoptysis.

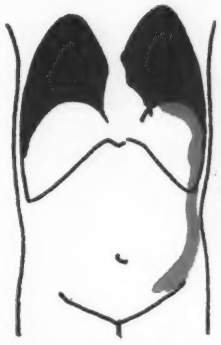
Sept. 13, 1947 : On admission he had a massive effusion on the right side with the mediastinum displaced to the left.



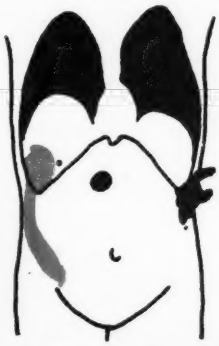
March 23, 1947 : Drainage of left perinephric abscess due to carbuncle of the kidney.



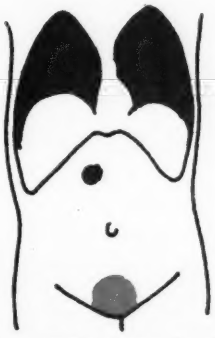
March 25 : Drainage of a left scrotal abscess.



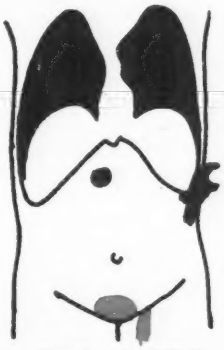
April 14 : Drainage of a very large subphrenic abscess tracking down the paracolic gutter. Nephrectomy.



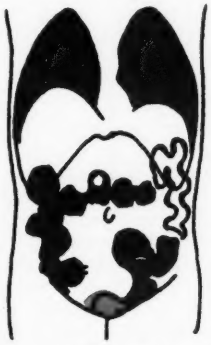
April 30 : Fæcal fistula formed at nephrectomy site. Drainage of right subphrenic abscess. Transverse colostomy done.



May 4 : Pelvic abscess forming.

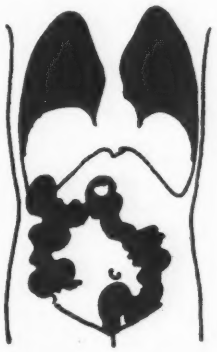


May 5 : Developed a left femoral thrombosis.



Aug. 21 : Exploration of the fæcal fistulæ and excision of splenic flexure and part of descending colon.

Sept. 4 : Pelvic abscess drained per rectum.



Jan. 28, 1948 : Anastomosis of the two blind ends of the large gut by the side-to-side method.

FIG. 279.—*Case* 8.

A triumphant conclusion to the persistent endeavour on the part of the surgeons, nursing staff, and patient.

Case 9.—**Right extraperitoneal subphrenic abscess** (*Fig.* 280).

Sapper A., aged 21.

Admitted to the Medical Division direct from leave with the following history : Three weeks ago, whilst at home, felt feverish, went to bed, and was treated as for malaria.

Liver enlarged to one finger below the costal margin. He looked ill, was short of breath, and had a troublesome cough. He had served two years in the Far East.

Sept. 14 : T. 102° ; pulse 150 ; respirations 50. W.B.C. 23,000. Chest was aspirated and a large volume of " tea coloured " fluid withdrawn. Colour proved to be due to bile. Sterile on culture.

Sept. 16 : Very ill indeed with a most distressing cough, with copious bile-stained sputum. Emetine course started.

Chest aspirated daily. Fluid became thicker, opaque, and a golden yellow colour. Radiography showed massive effusion with a tension pneumothorax and collapse of the lung, which contained an abscess cavity. Liver puncture negative.

Sept. 27: Pleural fluid found to be infected with *B. pyocyaneus*. Transferred to the Surgical Division.

Jan. 8: Pain in the right shoulder and right side of the chest. Friction rub at the right base. A radiograph showed a small pleural effusion but the diaphragm was not raised and was moving normally.

Jan. 12: Tenderness between the lower ribs at the back on the right side. At this stage a subphrenic abscess was diagnosed but the possibility of confirming the diagnosis and of localizing its position by the induction of a pneumoperitoneum was considered. A thousand c.c. of air were introduced and revealed a clear air space below the left diaphragm, which was raised to the level of the right (*Fig.* 282). No air was seen below the right diaphragm which had risen since the previous radiograph and was immobile (*Fig.* 283). Unfortunately the lateral view was unsatisfactory.

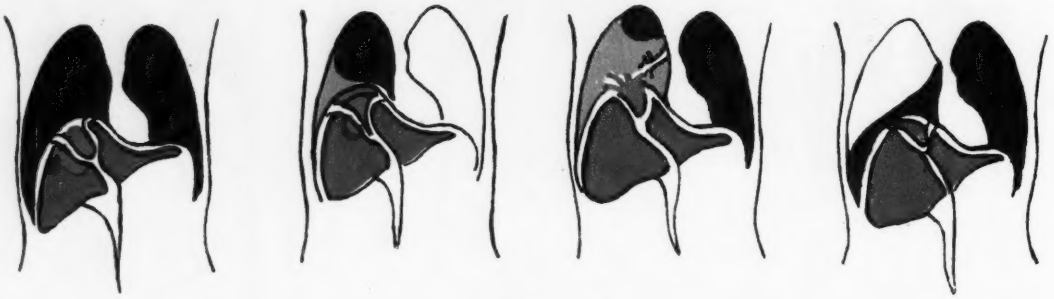


FIG. 280.—*Case* 9.

Jan. 13: A large right posterior and inferior subphrenic abscess was drained by resecting the 12th rib and the patient subsequently made a steady recovery.

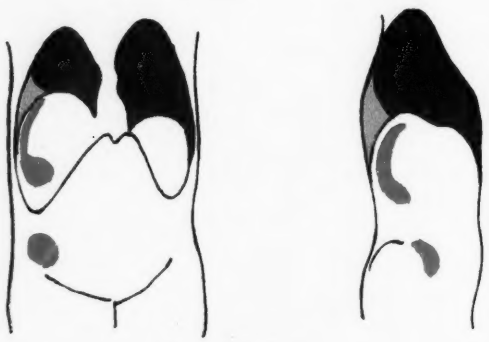


FIG. 281.—*Case* 10.

Sept. 29: Resection of the 11th rib and closed suction drainage. Drainage of 20 oz. per day with no general benefit. Emetine continued and supplemented by penicillin and sulpha drugs. Also repeated small blood transfusions. The only improvement was a slightly decreased volume of sputum.

Oct. 13: Right Kocher incision. Liver explored under direct vision. Abscess of the right lobe found ruptured into the 'bare area'. Drain brought out through the loin. Gradual recovery with less and less sputum, which finally became frank pus and then disappeared altogether.

Jan. 3, 1948: Discharged from hospital. There was a permanent collapse of the lung with firm adhesions limiting the movements of the diaphragm.

Comment.—One of the less common conditions predisposing to subphrenic abscess, but one which will be seen more often as the result of service overseas.

It is clear that the patient had a broncho-biliary fistula as well as a bronchopleural fistula, the most distressing feature from the patient's point of view being the extremely irritating nature of the bile in the bronchial tree. This irritation probably explains the massive effusion also.

It was hoped that draining the pleural cavity would be enough, but it is clear, looking back, that the second operation which provided *dependent drainage* was the real requirement.

The need for a full course of treatment for the primary condition, i.e., amœbiasis, must be remembered to ensure the best results and prevent recurrence.

The early diagnosis was missed.

Case 10.—**Right posterior subphrenic abscess** (*Fig.* 281).

Lieutenant B., Royal Navy, aged 22.

Admitted to this hospital off a ship. History as follows:—

Dec. 22, 1947: Acute midline abdominal pain, constant but varying in intensity. Later localized to the R.I.F. Fourteen hours after admission, appendicectomy via Battle's incision. Free fluid present. Drained. (No note of the peritoneum being sucked dry.) Drain removed in 48 hours. Two days later recurrence of abdominal pain with distension and vomiting.

Dec. 28: Admitted to this hospital with ileus. Treated by continuous gastric suction, intravenous drip, and morphine. Within 48 hours the condition had much improved.

Jan. 6, 1948: By this time an abscess had localized deep to the scar in the R.I.F. This was drained by a small transverse incision placed well out in the R.I.F.

Comment.—There is no note of how this patient was nursed in the early stages of the illness, but after arrival at this hospital Fowler's position was adopted. Some would say that this predisposed to the upward spread of infection. I believe that if infection is well localized this is not so, and that in selected cases it gives dependent drainage. It is just as likely that infection was established high up when the bowel was in a state of ileus and the diaphragm movements limited by bowel distension.

It is the diagnosis of subphrenic abscess in its *early* stages that is difficult and the induction of pneumoperitoneum was an attempt at seeing if this would help in future cases. It is possible that with experience early diagnosis and, more important still, accurate localization may be achieved. The dangers of this procedure are obvious and its value doubtful at present, and it should not be carried out without very careful thought.

One of the most interesting features of this case was the presence of a strain of *Actinomyces* in the culture of pus. It was sensitive to penicillin and as the patient has made a good recovery so far, it is unlikely, though still possible, that there will be any further complications.

Case 11.—**Right anterior subphrenic abscess** (*Fig.* 284).

Private B., aged 20.

Nov. 28, 1947: Admitted to the Medical Division with a two weeks' history of pain over the right costal margin and in the tip of the right shoulder, sharp and shooting, and made worse by deep breathing. Slight cough only.

During the next fourteen days he was kept under observation and ran a low-grade fever without any other abnormal physical signs.

Dec. 9: Temperature rose to 103° and W.B.C.s to 14,000. Continued to run a high fever and diagnosed as an amœbic hepatitis and treated with emetine. X-ray screening showed poor movement of the diaphragm, but no localized swelling. No evidence of amœbiasis was found in the stools.

Fever continued and the W.B.C.s rose to 30,000; a tender indurated mass appeared at the right costal margin.

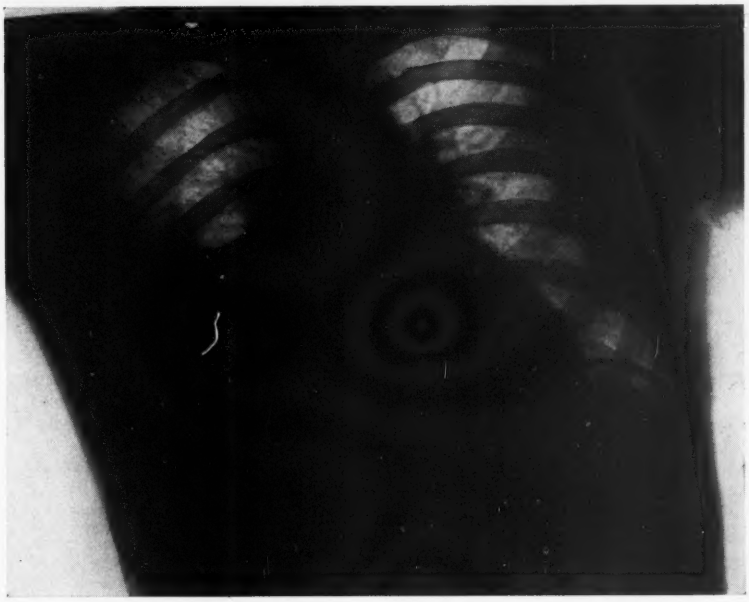


FIG. 282.—*Case* 10. Pneumoperitoneum in a case of right-sided subphrenic abscess, showing air under the left diaphragm but none under the right.

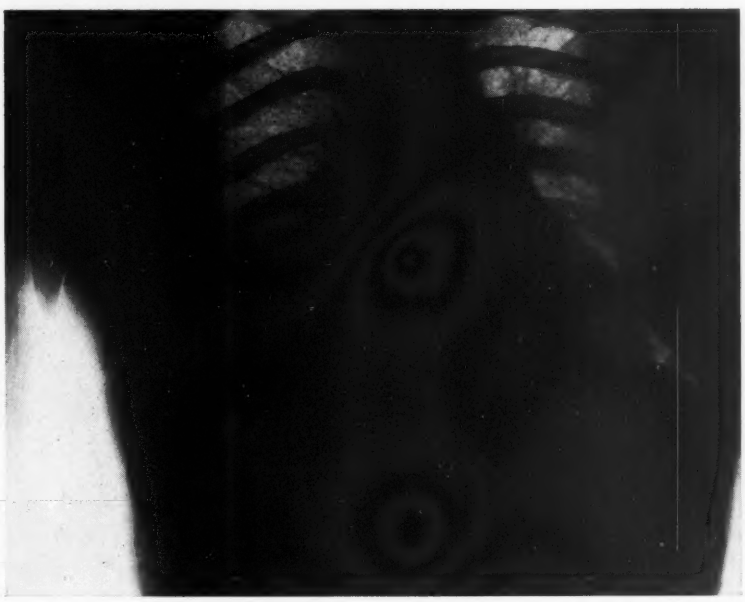


FIG. 283.—*Case* 10. Anteroposterior view showing raised right diaphragm with pleural effusion.

Dec. 22: Oblique subcostal muscle-cutting incision and an extraperitoneal approach to a large abscess above the dome of the liver. Much thick yellow pus evacuated. Culture: *Staph. aureus* and *B. coli*. Treated with penicillin 50,000 units six-hourly and sulphamezethine.

Operation was followed by an uninterrupted recovery.

Comment.—The patient had served overseas and the culture of the pus does not exclude the possibility

of this being an infected amœbic abscess. There is nothing in the history to suggest any other primary cause.

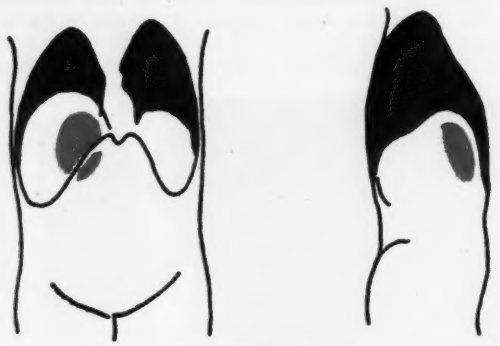


FIG. 284.—*Case* 11.

A point of interest in this case is the history that his father died of "an abscess above the liver".

Case 12.—**Left posterior subphrenic abscess** (*Fig.* 285).

Gunner T., aged 23.

Aug. 5, 1947: Knocked down by a lorry. Contusion of the right chest, left chest, and left shoulder. Shocked. Vomited gastric contents containing blood. Put on penicillin. P. 120; Hb 65 per cent; W.B.C.s 13,000. Remained ill, with high pulse-rate, copious vomiting, and increasing abdominal distension. Given small blood transfusions and gradually improved.

Aug. 16: Tender in the L.I.F. Temperature beginning to swing.

Aug. 20: Onset of mucous diarrhœa.

Aug. 23: Radiography—gross elevation of the left diaphragm.

Aug. 25: Suprapubic tenderness. ? fluid in peritoneum. W.B.C.s 10,600. Needle passed in the subphrenic space posteriorly. No pus or fluid withdrawn. Large tender swelling in the pelvis.

Sept. 1: Left lower paramedian incision. Large collection of blood-stained fluid in the pelvis. Adhesions more marked in left subcostal region. Pelvic collection drained. Notes state: "These findings suggest that there had been some bleeding a month ago, probably from a small tear in the spleen."

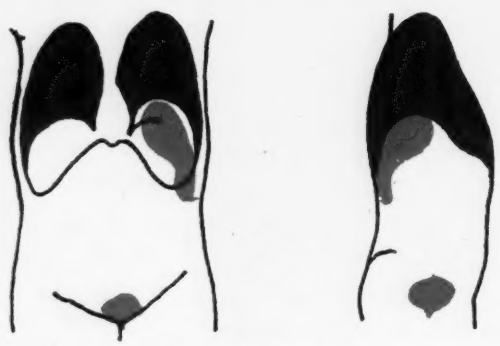


FIG. 285.—*Case* 12.

Sept. 13: General condition very poor. Pain in left chest and shoulder. Non-productive cough.

Sept. 23: Left diaphragm grossly raised. Drainage of subphrenic abscess by resection of 10th rib.

Sept. 30: Tube removed. Re-inserted Oct. 14 and finally removed Nov. 23. Subsequent uninterrupted recovery.

Comment.—It is doubtful if this was really due to rupture of the spleen. It would seem more reasonable to diagnose a small rupture of the stomach, especially in view of there being blood in the vomit.

It is difficult to explain infection of a splenic hæmatoma unless this was introduced at the time of 'blind needling'. The need to re-insert a drainage tube frequently arises if an abscess has remained undiagnosed for a long time and also if the drain has been placed too high. Under these circumstances there is an 'overflow' drainage and quite often the tube has to be re-inserted through a new incision lower down.

It is quite possible to diagnose a subphrenic abscess as an empyema, especially if the patient has been 'chesty' after operation. If this is so, too high a drainage may be done.

Reading through the notes, it is almost certain that infection was well established under the diaphragm as far back as Aug. 23.

CONCLUSIONS

Subphrenic abscess may follow inflammation in almost any intra-abdominal organ and must be borne in mind.

FIG. 286.—Anteroposterior view of a fluid-containing abscess cavity. Is the tube in the cavity and is it in the most dependent part?

Early and efficient treatment of any predisposing cause such as appendicitis or peptic ulcer will tend to prevent it.

Any predisposing cause must be treated throughout in order to get the best results and prevent relapses.

The controversy over Fowler's position in the nursing of patients and the theories of the spread of infection from below upwards by the lymphatics and capillary attraction make it difficult to decide on the best methods of prevention.

Fowler's position is difficult to maintain and uncomfortable for the patient and tends to restrict

the breathing, but there can be little doubt that on occasions it affords the best method of getting dependent drainage.

In a diffuse inflammation it is unlikely that a drainage-tube will drain anything beyond its immediate track after the first 48 hours and should therefore be removed.

On the other hand, in a well-localized case, such as an abscess, it will continue to do so as long as there is anything to drain or until the abscess walls collapse and the abscess cavity becomes obliterated.

It is in such cases that Fowler's position may be employed with advantage.

Early diagnosis is difficult and frequently missed. Service cases often suffer from the disadvantage of having to pass through many hands, with the inevitable delay in diagnosis and treatment.

As a rule a subphrenic abscess needs drainage as soon as a diagnosis is made.

Diagnostic needling should be avoided. The dangers of spreading infection to pleura or peritoneum or of damaging some intra-abdominal organ are real.

The longer a subphrenic abscess remains untreated the more likely it is that chest complications will follow and the chances of recovery diminish.

If radiographs show a fluid level, the size, shape, and most dependent part of an abscess cavity may be visualized and subsequently the best surgical approach and nursing position decided upon (*Figs.* 286–288).

The extra-peritoneal approach should always be employed. An anterior subcostal or paramedian incision is suitable for the anterior spaces and resection of the 12th rib for the posterior spaces and the right inferior space.

Great care is needed to avoid injury to the splenic flexure in left-sided cases.

Many various types of organism may be found on culture of the pus. Massive doses of penicillin supplemented by the sulpha drugs are beneficial.

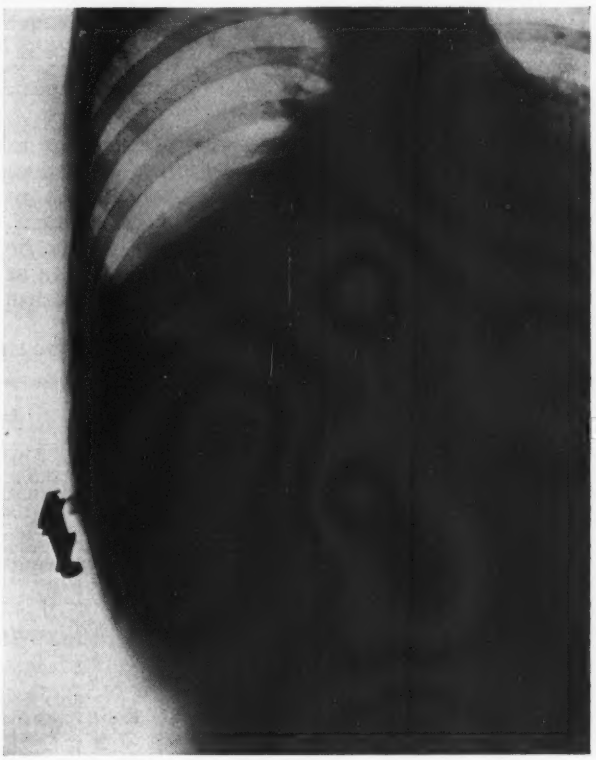


FIG. 287.—Anteroposterior view of the same case as in *Fig.* 286, taken with the patient lying on his left side. Shows the tube well within the cavity. Also outlines the lower limits of the cavity.

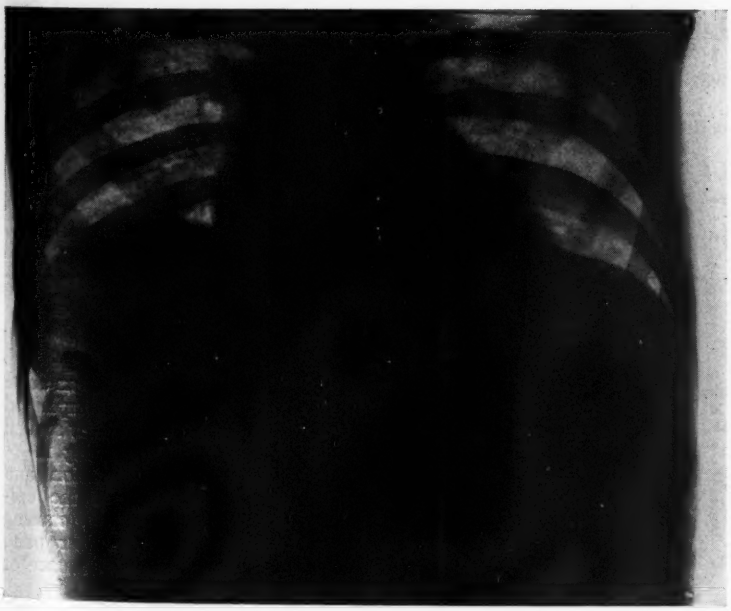


FIG. 288.—Anteroposterior view showing a stomach bubble. Not to be confused with a gas-containing abscess cavity.

The patient's natural resistance may overcome the remainder that do not respond to this therapy.

A nourishing diet of high protein value, breathing exercises from the start, and careful nursing all help to hasten recovery.

I am inclined to think that the anatomical descriptions of the subphrenic spaces are of academic rather than practical interest.

Infection may be localized in one of them but is confined as much by freshly formed adhesions as it is by the ligaments of the liver and folds of peritoneum. Infection seldom corresponds with the anatomical boundaries of a space, sometimes occupying part of a space and frequently filling more than one space.

I am grateful to Col. A. R. Ross, R.A.M.C., for permission to publish these cases, and to Brigadier D. Fettes, Consultant Surgeon to The Army, and Lieut.-Col. D. Bowie, R.A.M.C., for their help and encouragement.

MASSIVE SPONTANEOUS INTRAPERITONEAL HÆMORRHAGE

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MASSIVE spontaneous intraperitoneal hæmorrhage in the male is a rare condition. The term 'abdominal apoplexy' denotes the close resemblance both in spontaneity and nature to the much more frequent, and better known, cerebral apoplexy. It may be impossible to find the cause of the hæmorrhage at operation. In other cases (in either sex) the cause is usual to exclude these obvious pathological and traumatic conditions, also hæmorrhage from rupture of a large aneurysm, when referring to massive spontaneous intraperitoneal hæmorrhage. A study of the literature has revealed 28 cases (*Table I*), which we have tabulated, and to which we have added 1 of our own.

Table I.—ESSENTIAL DATA IN REPORTED CASES OF INTRA-ABDOMINAL APOPLEXY

CASE NO.	AUTHOR	YEAR	SEX	AGE	LOCATION	C.V.S.	OPERATION	RESULT	P.M.
1	Barber	1909	F	32	N.D.B.P.	Normal	Yes	Recovered	—
2	Churchman	1911	M	48	N.D.B.P.	Normal	Yes	Died	No
3	Florence and Ducuing	1913	F	?	Sup. mesen. A.		No	Died	Yes
4	Hilliard	1918	M	48	N.D.B.P.	Hypertension	Yes	Died	No
5	Starcke	1923	M	60	Gastro-duod. A.	Hypertension	Yes	Recovered	—
6	Budde	1925	M	27	L. gastro-epiploic A.	Normal	Yes	Recovered	—
7	Green and Powers	1931	F	54	L. gastric A.	Hypertension	Yes	Recovered	—
8	Morgue-Molines and Cabanac	1933	M	56	L. gastric A.	?	Yes	Recovered	—
9		1933	?	?	L. gastric A.	Arteriosclerosis and hypertension	Yes	Recovered	—
10		1933	F	73	L. gastro-epiploic A.	Marked arterio-sclerosis	Yes	Died	Yes
11	Hartley and MacKechnie	1934	M	31	N.D.B.P.	App. normal	Yes	Died	Yes
12	Thompson and Dunphy	1935	F	62	L. gastric A.	Hypertension	Yes	Recovered	
13	Buchbinder and Greene	1935	M	57	R. gastric A.	Hypertension	Yes	Recovered	
14	Moorehead and McLester	1936	M	44	Junction R. and L. gastric A.	Hypertension	No	Died	Yes
15		1936	M	50	Aneurysm of sup. mesent. A.	Hypertension and arteriosclerosis	No	Died	Yes
16	Bruce	1937	M	34	N.D.B.P.	Normal	Yes	Recovered	—
17		1937	M	75	Aneurysm mid. colic A.	Abd. vessels normal	—	Died	—
18	Nevin and Williams	1937	F	45	Cong. aneurysm of splenic artery. Cong. aneurysm of brain.	Hypertension	No	Died	Yes
19		1937	F	56	N.D.B.P.	Rupt. cong. aneurysm of brain	No	Died	Yes
20	Morton	1938	M	72	Sup. mes. A.	?	Yes	Recovered	
21	Silverstone	1938	M	52	N.D.B.P.	Hypertension	Yes	Died	
22	Berk, Rothschild and Doane	1939	M	52	N.D.B.P.	Hypertension and arteriosclerosis	Yes	Recovered	
23	Madden and Bunch	1941	M	62	Sup. mesenteric A.	Arteriosclerosis	Yes	Recovered	
24	Crile and Newell	1940	M	49	Mid. colic A.	Hypertension	Yes	Recovered	
25	Lafferty and Pearson	1940	F	28	Mid. colic A.		No	Died	Yes
26	Shallow and Herbut	1946	M	50	Aneurysm of inf. pancreatico-duodenal A.	App. normal	No	Died	Yes
27	Sorrel and Alison	1945	?	4	N.D.B.P.	App. normal	Yes	Died	Yes
28	Reiman and Cowley	1946	M	44	Luetic aneurysm of cœliac axis	No	Yes	Died	Yes

N.D.B.P.—No definite bleeding point

may be obvious—such as gross visceral disease; eroding primary or secondary tumours of the liver, spleen, pancreas, kidney, or ovaries; pathological rupture of a viscus; or pancreatitis. Bruce (1937) states: "A variety of conditions that are common to the two sexes may manifest their presence by severe intra-abdominal hæmorrhage and their nature is often obvious at operation". This is true of spontaneous rupture of the spleen (Bailey, 1929–30) and liver (Bruce, 1929), rupture of splenic vessels in cirrhosis of the liver (Pyrah and others, 1929; Ogilvie, 1922), oozing from the liver in toxic hepatitis (Peck, 1905), in undulant fever (Box and Bamforth, 1925), and ruptured mycotic aneurysm in acute bacterial endocarditis (Willius, 1935). However, it

CASE REPORT

A. M., a clerk aged 39 years, was admitted as an acute abdominal emergency on Oct. 25, 1947. He complained of severe epigastric pain of 30 hours' duration. For several years he had had a mild indigestion commencing 10–15 minutes after food. There had been remission and exacerbation. The present attack started in the epigastrium and gradually became more severe, causing generalized abdominal pain, with right shoulder pain. The appetite was not lost, and there was no nausea or vomiting. Recently the bowels had become constipated. He had never had melæna. Apart from asthma and bronchitis in the winter, there was nothing significant in the past history.

PHYSICAL EXAMINATION.—The characteristic findings were marked pallor of the skin and mucous membranes.

The abdomen was slightly distended and was silent. Shifting dullness was not elicited. There was no abdominal tenderness or rigidity. A few scattered rhonchi were present in both bases. The other systems were normal. The blood-pressure was 100/70. Temperature 97·4°; pulse 72; respirations 22.

A provisional diagnosis of a bleeding peptic ulcer was made and the patient was left under observation. Four hours later the pulse-rate increased to 100 per min., without a corresponding rise in temperature. The blood-pressure was now 80/40. An indefinite swelling was palpable in the right iliac fossa, and there was an increase in the lower abdominal distension. As the patient's condition was deteriorating, it was decided to explore the abdomen.

AT OPERATION.—Open ether was used as anæsthetic. A small split-muscle incision was made in the right iliac fossa.

The peritoneum, blue in colour, bulged into the wound. A blood transfusion was commenced and the peritoneal cavity opened. About two pints of blood was aspirated from the peritoneal cavity. The lower abdomen and pelvis was explored, but no bleeding point was found. A midline supra-umbilical incision was made. The lesser sac was seen bulging with blood. It was opened. About one pint of blood clot was scooped from the lesser sac. No bleeding vessel or perforation was found in the stomach or duodenum. The transverse mesocolon and omentum showed no hæmatoma. The tail of the pancreas was slightly swollen and hæmorrhagic, but the peritoneum in this situation was not torn. The spleen and its vessels were visualized and, although there was blood around the spleen, no rupture was found. The liver and its vessels, the small intestine, and the mesentery were normal. There was no Meckel's diverticulum and no fat necrosis. The wound was closed in layers and a split tube and wick drain inserted through the wound. After four pints of blood had been given, the transfusion was discontinued. Apart from a transient jaundice, the convalescence was uneventful. Patient discharged on the fourteeth day.

INVESTIGATIONS.—Prothrombin index normal. Urinary diastase per 24 hours—19,250 units. W.R., negative. Widal test, negative. On discharge the blood-count was normal.

Table II.

	Cases
Arteriosclerosis and hypertension	14
Normal arteries	8
Doubtful	6
No definite bleeding point	9
N.D.B.P. and hypertension	3

DISCUSSION

This is a case of massive spontaneous hæmoperitoneum. There was no evidence of arteriosclerosis, aneurysm, or trauma. Although the patient was subject to asthma and bronchitis, he had been free of these conditions for about six months. Churchman (1911) regarded his case as being hæmophilic, largely because intraperitoneal hæmorrhages are known to occur in fatal cases of this disease and in experimentally produced 'hæmorrhagic diseases' (Whipple and Sperry, 1909). However, Osler and McCrae (1925) regard Churchman's case as an acute form of 'hæmorrhagic peritonitis'. Sorrel and Alison's case (1945) is interesting as the child died four days after operation, and typhoid ulceration was found in the intestine. They do not mention whether the bleeding resulted from the ulceration. In the 28 cases traced in the literature, 14 showed definite evidence of arteriosclerosis or hypertension, 8 were normal, and 6 were doubtful.

There are only 8 cases in the literature of massive spontaneous hæmoperitoneum in the absence of hypertension and arteriosclerosis (Barber, 1909; Churchman, 1911; Hartley and MacKechnie, 1934; Bruce, 1937; Nevin and Williams, 1937; Sorrel and Alison, 1945; and Shallow and Herbut, 1946).

Arteriosclerosis is the commonest cause. In the patients with arteriosclerosis and hypertension, the bleeding vessel can usually be found at operation as the artery is unable to contract. Ligature of the bleeding point results in cure and, surprisingly, no recurrence of the bleeding in these cases has yet been reported.

The position is, however, different in younger patients or where no obvious bleeding point is found in an apparently normal cardiovascular system. The heart is incapable of raising the blood-pressure sufficiently high to cause rupture of a normal artery, and some congenital or developmental defect of the artery must precede spontaneous rupture.

Only 2 cases which fall into this category have recovered (Barber, 1909; Bruce, 1937). The reported case herewith makes the third. Bruce (1937) suggested that the hæmorrhage in this group may be due to rupture of a miliary aneurysm or at a 'junctional area'. Some support is given to this by the record of two patients who had massive intraperitoneal hæmorrhage from an aneurysm in the middle colic artery (Illingworth, quoted by Bruce, 1937), and from an aneurysm of the left gastro-epiploic artery (Budde, 1925). In both cases there was no evidence of arteriosclerosis.

Bruce's suggestion was soon confirmed by Schuster (1937), who described a case of multiple aneurysms of the splenic artery which she believed were of congenital or developmental origin in a case of familial hæmorrhagic telangiectasis. Nevin and Williams (1937) described 2 cases of massive intraperitoneal hæmorrhage. One was due to rupture of a congenital aneurysm of the splenic artery associated with a second rupture of one out of four congenital aneurysms present in the cerebral arteries. In the second case no definite bleeding point was found in the abdomen, but an unruptured congenital aneurysm of the cerebral arteries was found at autopsy.

Finally, Shallow, Herbut, and Wagner (1946) demonstrated defects in the muscle coat of the inferior pancreatico-duodenal artery, associated with a ruptured congenital saccular aneurysm at the first bifurcation and 3 small fusiform dilatations in the more distant part. They believe originally there was a congenital or developmental defect along the course of the artery consisting of what appeared to be a complete or partial failure of fusion of the media. At first, this defect was most likely covered by the internal elastic membrane and its intima, but as the vessel was stretched, these coats were broken, leaving in some places the adventitia alone to form the entire remaining wall.

No reference to periarteritis nodosa showing spontaneous massive hæmoperitoneum has been found.

The diagnosis of massive spontaneous intraperitoneal hæmorrhage is usually made at laparotomy or autopsy. When confronted with this condition in the male, paracentesis will differentiate it from severe internal hæmorrhage in the gastro-intestinal tract. If operation is performed, a lower abdominal incision will be the best in the female, as the commonest cause of intraperitoneal hæmorrhage is ectopic pregnancy, other lesions of the ovary, and ruptured uterine veins.

In the male, an upper midline incision should be used, as this allows adequate exposure of the liver and spleen and their vessels. When these organs are not the cause, the mesenteric and gastric vessels are carefully examined for the bleeding point, ligature of which is a life-saving measure. Finally, the diaphragm is examined for bleeding from a ruptured varicose vein.

SUMMARY

1. Twenty-eight cases of massive spontaneous hæmoperitoneum have been tabulated.

2. The history and progress of a further case which recovered after laparotomy is given.

3. The aetiology is discussed.

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PULMONARY RESECTION FOR METASTATIC MALIGNANCY

THREE CASES SECONDARY TO HYPERNEPHROMA

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A RECENT review of 5 recorded cases and 19 additional cases by Alexander and Haight, prompts the recording of the 3 following cases, in each of which the primary tumour was a hypernephroma.

CASE REPORTS

Case 1.—

HISTORY.—Mrs. A. F. C., aged 42, was seen first at Green Lane Hospital Thoracic Unit on April 5, 1945, having been referred by Dr. Russell Nolan of the Radiotherapy Department, Auckland Hospital.

In February, 1933, left-sided nephrectomy had been done by Mr. Frank Macky for hypernephroma. The tumour was the size of a grape-fruit and occupied the upper pole. Hæmaturia had been present off and on for four years before that admission, so it is likely that the tumour had been present even in 1929. Microscopically the growth was an adenocarcinoma.

In 1941, cough, sputum, and fever developed, and a film on Sept. 5 showed an oval shadow 5·5 × 4·5 cm. in the upper part of the lower lobe of the left lung. It had fairly sharp outlines, and was of even density. It was regarded as a metastasis from the hypernephroma. No others have been discovered in lung fields or bones despite repeated searches up to the present time (September, 1947).

X-ray therapy was applied on three occasions: in September–October, 1941 (4600 r), August, 1942 (4500 r), and June, 1944 (3750 r). The shadow diminished markedly in size but never disappeared, and by April, 1945, it measured 8 × 7 cm. Dr. Nolan was reluctant to give any more therapy. Bronchoscopy was not carried out.

AT OPERATION.—Exploration was agreed to and carried out on April 28 through the bed of the resected seventh left rib. A good view was obtained. There were very few adhesions. The spherical growth was found peripherally placed in the upper part of the lower lobe. The hilar region was normal. The interlobar fissure was developed readily. At one point, as the separation was being made, a hole appeared in the growth and friable tissue began to squeeze out. A little was spilt. No residual infiltration was left on the lower face of the upper lobe. The remainder of the lobectomy was completed easily by hilar dissection.

PROGRESS.—Convalescence was delayed by empyema and trouble with some non-absorbable sutures, but was

steady and complete. Up to date, two and a half years after lobectomy, no sign of recurrence of the growth in the chest or elsewhere has been discovered.

THE SPECIMEN (Dr. W. Gilmour's report).—A tumour 5 × 5 × 4 cm. in the substance of the lower lobe, extending from the hilum to near the anterior border, and ruptured through the pleura anteriorly. It was soft, friable, and uniform in appearance, not hæmorrhagic. Microscopically the structure was papillary, the papillæ being mostly lined by a single layer of cells. There was some infiltration of the stroma, but the tumour appeared to have a very low grade of malignancy. It was probably metastatic from the renal tumour.

Case 2.—

HISTORY.—Mr. C. M. M., 63 years, was seen first at Green Lane Hospital Chest Clinic on Aug. 23, 1946, on account of three months' cough and sputum. The whitish sputum had been blood-stained in the last four weeks. There was no loss of weight and the patient felt well.

Radiologically a circular shadow 3·5 cm. in diameter was seen in the left lung field near the border of the heart. No other abnormal shadows were found in lung fields or bony skeleton.

In 1940, after painless hæmaturia, a right-sided nephrectomy had been done in Thames Hospital by Mr. Edward Gifford for cancer. The tumour was a characteristic hypernephroma to the naked eye, being an adenocarcinoma microscopically.

Bronchoscopically (Dr. C. McDowell) there was no encroachment of the tumour on the bronchial tree. Two dorsal bronchi were seen leading to the right lower lobe.

AT OPERATION.—Exploratory thoracotomy and left pneumonectomy were carried out on Sept. 11, 1946. The mass was identified in the lower lobe at a little distance from the hilum, and lower lobectomy was carried out very easily on account of the depth of the fissure and the absence of adhesions. On section it was an encapsuled growth, partly yellow, partly necrotic, and was regarded as metastatic from the hypernephroma. Pneumonectomy was completed by high division of the main hilar structures and removal of the upper lobe for this reason, and because of two small nodules in the anterior border of the upper lobe, which were found to have the same appearance.

Microscopically the lung tumour was a "columnar-celled carcinoma with in places a papillary structure—probably a metastatic hypernephroma".

PROGRESS.—The post-operative course was uneventful and he remained well for eleven months save for one month in hospital, eight months after operation, for anterior myocardial infarction (Dr. E. H. Roche), from which he made a good recovery. At eleven months he began to go downhill with nodular enlargement of the liver, and general cachexia, almost certainly due to general widespread recurrence of the growth. He died on Nov. 27, 1947, fourteen and a half months after pneumonectomy. Post-mortem examination revealed multiple metastases from hypernephroma involving the local site of the original tumour, the inferior vena cava, the diaphragm, the chest wall, and a nodule or two in the other lung, amongst other places.

Case 3.—

Mrs. M. M., 65 years, was sent to Green Lane Hospital Chest Unit in December, 1947, as ? gastric ulcer with hæmatemesis. The latter proved to be hæmoptysis. There had been a wheeze and a cough, with brownish sputum, for three months. She had been dyspnœic on exertion, and this was probably related to hypertension (180/100 on admission), for which she had been under treatment for four years. She was said to have lost 6 lb. in the previous three weeks, but was still stout.

HISTORY.—Included right nephrectomy by Mr. Frank Macky in March, 1938, for a bulky hypernephroma 5 in. long occupying the middle part of the kidney. The structures of the pedicle were not clearly displayed. Convalescence was uneventful. She said she had known of a lump in her right upper abdomen for ten years. It had slowly increased in size, and one minor hæmaturia only had precipitated her admission. She had reported this lump when in hospital in 1928 for appendicectomy, but though it was alleged to be "the size of a mandarin orange and not appearing to be connected with the kidney", no tumour was found at the operation—only an enlarged liver.

ON EXAMINATION.—Radiologically showed atelectasis of the left lower lobe to be almost complete. No outline of a tumour could be made out. The right lung field was clear. Bronchoscopically (Dr. J. R. Hinds) the tree was normal on the right side, and also on the left as far down as the first dorsal branch to the lower lobe. Beyond this there was much bleeding and hæmorrhagic mucosa, from which a biopsy was taken. No tumour or other characteristic tissue was recognized.

AT OPERATION.—Thoracotomy was advised and carried out by Rowan Nicks and Douglas Robb, under intratracheal cyclopropane and oxygen by Dr. Alexandra Warnock. There were no adhesions. Most of the lower lobe was atelectatic, and an egg-shaped tumour was felt in the hilum of the lower lobe. It appeared well encapsuled. There were no glands around the hilum. Pneumonectomy proceeded without incident, and post-operative condition and course were uneventful.

THE SPECIMEN showed a normal tree down to just beyond the normal orifice of the first dorsal to the lower lobe. Beyond this point the mucosa of most of the bronchi was infiltrated with extension of the surrounding growth, and the surface was breached in parts. One bronchus was completely occluded by external pressure and was bronchiectatic beyond. The growth itself was yellow and hæmorrhagic in colour and microscopically of hypernephroma type. It appeared to have been completely removed. It had encroached on the wall of the lower pulmonary vein at two points and bulged into the lumen without breaking the intima. A small pea nodule of growth was found near the edge of the lower lobe.

Summary.—A third case, without follow-up story, of pulmonary resection for metastatic hypernephroma is recorded. The nephrectomy preceded the pneumonectomy by almost ten years, and according to the patient the abdominal tumour had been present for ten years before the nephrectomy. The tumour was oval and the size of a hen's egg, and differed from the other two recorded above in encroaching on bronchial lumen, causing hæmoptysis, atelectasis, and bronchiectasis.

PATHOLOGICAL REPORT (J. L. Pinnegar).—Macroscopically the specimen consists of a lung measuring in its fixed state 15 × 15 × 6 cm. approximately. At the hilum there is a large neoplastic mass, the extrapulmonary measurements of which are 3·5 × 2·5 × 5 cm. It is soft, pale brown, and bears small dark-brown areas. The tumour lies between the pulmonary vein and the bronchus, and extends below the vein to 3 cm. above the diaphragmatic surface of the lung, and above the vein is a point 6 cm. below the apex of the lung.

The vein itself shows two small nodules on the intimal surface, which are due to the tumour. The intima has not been invaded. The main lower lobe bronchus has been infiltrated, and the tumour has breached the mucosa over a considerable area.

Another tumour is present, 1·5 cm. in diameter on the other side of the lower lobe bronchus from the large mass mentioned above. This may be part of this large mass, but the lung has not been incised to determine this point.

A further small nodule was present approximately 1 cm. in diameter on the diaphragmatic surface 3 cm. from the posterior border, and having the same appearance as the main mass.

Other minute ill-defined nodules could also be palpated. On section these were dark brown.

Histology.—

1. The Main Mass: Secondary carcinoma, which is separated from the collapsed and congested lung surrounding it by a thin rim of fibrous tissue for most of its extent. The cells nearly all have clear vacuolated cytoplasm. The picture would do very well for secondary hypernephroma.

2. Tumour on the Diaphragmatic Surface of the Lung: Secondary carcinoma of similar structure.

3. One of the Minute Dark-brown Nodules mentioned above: Secondary carcinoma of similar structure.

COMMENTS

Concerning *pulmonary metastases in general*, Alexander and Haight give the following list of primary sources in their 24 cases:—

Sarcoma (various)	8
Renal cell carcinoma	6
Rectum	3
Sigmoid	1
Colon	1
Ovary	1
Uterus	1
Bladder	1
Mandible	1
Testis	1
Total	24

Post-operative survival is given by them as follows: In 12 cases 11 had recurrence and 1 died after pneumonectomy. In the remaining 12 cases there had been no recurrence after 12, 7, 6, 5, 4, 3, 1½, and 1 year after pulmonary resection, plus 4 recent cases. The sarcomata did better than carcinomata, and renal-celled tumours also did well. This is satisfactory, as these two groups supplied the majority of the cases.

Concerning *hypernephroma in particular*, our *Case* 1 almost equals the classical case of Barney and Churchill, which in 1944 had completed 12 years of survival after nephrectomy. Although the metastasis in the lung was the first evidence of the tumour, it was not until fifteen months after the nephrectomy that the lobectomy was carried out. Our *Case* 1 has remained well two and a half years after lobectomy, fourteen and a half years after nephrectomy, and eighteen and a half years since the first probable clinical evidence of the hypernephroma.

The hypernephroma, or renal adenocarcinoma or Grawitz tumour, is an interesting growth from many points of view. It has a marked tendency to metastasize to lung and bone, often after long intervals of apparent surgical cure of the primary (as in the present cases, eight, six, and ten years respectively). Other features are the tendency of these tumours to cause pyrexia, the frequency with which the primary growth is 'latent' (R. A. Willis), revealing itself at long or short intervals after a metastasis has been found (as in Barney's case), and the occurrence of spontaneous regression of pulmonary metastases (Beer), even when they are multiple and widespread, and when no treatment of any sort has been applied. Not only lung and other distant metastases, but also local ones occur, both early and as late as seven to ten years afterwards, and X-ray therapy to the primary site on the original occasion has no apparent influence on their subsequent appearance or non-appearance. Beer remarks that the late metastases tend to be solitary, so that we may possibly achieve better results with pulmonary resection where the interval between it and the nephrectomy is a long one. Barney's case, however, is an example to the contrary.

Clearly there are many biological features unexplained in connexion with the appearance, growth, and resolution of these erratic tumours. Pending a more complete understanding, and in view of the clinical experiences described, the following practical measures are suggested for the guidance of general and thoracic surgeons and urologists:—

1. Chest films are an important feature of the follow-up examinations in hypernephroma, and should be continued every three to six months up to ten years after nephrectomy, if not longer.

2. Pneumonectomy or lobectomy is worth doing for solitary metastasis, and would seem to be more promising the later it is called for. When the growth is limited to one lobe, is peripherally placed, and is well encapsuled, lobectomy appears to give excellent results.

3. A single spherical opacity in the lung field, often not accessible to the bronchoscope, may be the first sign of a hypernephroma. Repeated clinical and urographic studies may be necessary before the primary is demonstrated. In the present state of knowledge, the pulmonary growth should probably be dealt with surgically at once.

4. X-ray therapy failed to control the pulmonary metastasis in Barney's case. In our *Case* 1, however, it seemed to hold it in check for four years, though just before lobectomy its dimensions were larger than they had ever been.

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SPONTANEOUS RUPTURE OF THE SPLENIC ARTERY

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THE splenic artery is peculiarly liable to aneurysm formation. Such aneurysms may rupture into the stomach, colon, retroperitoneal tissues, or peritoneal cavity. The inherent weakness of the arterial wall may also lead to spontaneous rupture without previous aneurysm formation, as in the cases described by Smith (1911) and by Macleod and Maurice (1940). Of the following cases, the second undoubtedly falls into the latter group, and it is probable that the first should also be included, although the presence of an aneurysm was assumed when it was previously described. A disproportionate number of the reported cases of intraperitoneal rupture have occurred during the later months of pregnancy and the accident occurs in healthy adults with no evidence of generalized arterial degeneration, syphilis, or other predisposing cause. The hæmorrhage is liable to take place in two attacks, the first resulting in a hæmatoma in the lesser sac and the second usually proving rapidly fatal. Of my two cases the first was fortunate in that the second hæmorrhage did not prove immediately fatal, time being allowed for splenectomy to result in a cure. The second case probably owed his life to the experience gained from the first. He presented at operation as a spontaneous hæmoperitoneum; the hæmorrhage had ceased, but the fact that the lesser sac was full of blood led me to remove the spleen; a small hole in the branch of the splenic artery was found after the operation.

FIG. 289.—Section of the ruptured artery.

FIG. 290.—Section of normal branch of similar calibre.

CASE REPORT

Case 1 (previously reported, Gillam, 1942).—A healthy woman, aged 26, in the eighth month of pregnancy, was admitted to the hospital about twelve hours after the onset of acute abdominal pain. She was suffering from shock. The abdomen was tender all over and slightly resistant. The uterus felt tense and fœtal heart sounds were inaudible. A diagnosis of concealed accidental hæmorrhage or acute hydramnios was made and labour was induced by rupturing membranes. After delivery of a stillborn fœtus her general condition remained poor and epigastric distension and vomiting was thought to be due to acute dilatation of the stomach, for which appropriate treatment was given. She rapidly recovered and was discharged from hospital a fortnight later in good health and free from any symptoms or signs of abdominal disease.

The next morning she was again seized with severe abdominal pain and returned to hospital severely shocked. After resuscitation, laparotomy was performed. The general peritoneal cavity was full of blood. After this had been evacuated a mass of blood-clot was felt in the lesser sac below the spleen. The lesser sac was opened and after removing the clot the hæmorrhage from the splenic pedicle was so severe and, owing to the presence of old adherent clot, so difficult to locate accurately that the spleen had to be hastily removed and no search of the ligatured pedicle was made to determine whether an aneurysm was present.

She made a good recovery and when seen a year later had passed into the W.A.A.F.

Case 2.—A previously healthy airman aged 28 was seized with epigastric pain during an operational flight. The plane had not risen above 3000 ft. and he was not conscious of any occurrence that could have produced any injury. He attributed the pain to a breakfast of spam. On admission to hospital five hours later he was suffering from shock. Abdominal pain was maximal in the epigastrium. He had vomited and was retching violently. The abdomen was slightly distended, tender all over, and slightly rigid. After resuscitation laparotomy was performed. The general peritoneal cavity contained a considerable quantity of blood. After this had been evacuated it became evident that the lesser sac contained blood and clots and was overflowing through the foramen of Winslow. After opening the lesser sac and removing the blood and most of the clots no bleeding point was found, but the conditions were so similar to those found in *Case* 1 that splenectomy was performed. He made a normal recovery and resumed duty a few months later.

The spleen looked normal but on removing clot from pedicle near the hilum a hole was revealed in a branch of the splenic artery.

The specimen was sent to Dr. A. F. Sladden who reports as follows:—

"The spleen is of normal size and texture with signs of a few adhesions along the inferior margin. There were no infarctions nor were any signs found of thrombosis

or other abnormality in microscopic sections of the organ.

"At the root of the spleen there was a mass of blood-clot of recent formation; this was derived from a rupture of one of the larger branches of the splenic artery about half an inch from its point of entry into the splenic tissue. To the naked eye there was no appearance of aneurysm at the site of the arterial rupture, but sections made through this part of the artery show much thinning of the medial wall (*Fig.* 289); this is very well seen when a section across another branch of the splenic artery is made for comparison (*Fig.* 290). No inflammatory reaction was found in the arterial tissues.

"It seems probable that there was a 'weak spot' in this branch of the splenic artery, probably congenital, and that the combination of adhesions along the margin of the spleen and this weakened wall led to rupture of the artery either because of some sudden movement or pressure effect upon the abdomen."

DISCUSSION

Sherlock and Learmonth (1942), who found an aneurysm of the splenic artery after removing the spleen in a case of Gaucher's disease, review the literature and comment on the frequent occurrence of aneurysm in the splenic artery. In discussing the possible aetiology they mention arterial degeneration, syphilis (very rare), splenomegaly, trauma, increased portal pressure, and congenital defects of the arterial walls, either intimal, muscular, or elastic tissue being deficient. In my two cases, and in those described by Macleod and Maurice (1940) and others, the patients were young and healthy, free from arterial disease, splenomegaly, and syphilis. It seems probable, therefore, that their pathology was similar to that described by Dr. Sladden above, and that the splenic artery is liable to have weak spots, perhaps congenital in origin, where rupture with or without previous aneurysm formation is prone to occur.

From the surgeon's viewpoint it is desirable that the condition should be familiar as the occurrence of a spontaneous hæmoperitoneum in the absence of ectopic gestation or ruptured spleen may prove a difficult problem, more especially if, as in *Case* 2, the hæmorrhage has ceased. If, however, there are blood and clots in the lesser sac it is highly probable that the hæmorrhage is from the splenic artery or one of its branches.

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DUODENOCOLIC FISTULA COMPLICATING CARCINOMA COLI

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FISTULA formation is a recognized though uncommon complication of cancer of the digestive tract, the site of the fistula being usually between the stomach and the colon. Fistulous communication between the duodenum and colon is a much rarer occurrence.

The great majority of reported cases of duodenocolic fistula have, as might be expected, been autopsy material. In the past twelve years, apart from the reports of Mindline and Rosenheim (1935) and Saleh (1935), there have been no instances recorded in this country, and the former authors could find no previous case in the literature. The condition has been described from other sources—Carnot and Caroli (1936), Fossati (1938), Hubeny and Delano (1940), Feldman (1942), and Linton (1944). In addition to these communications McPeak (1940) and McClinton (1944) have both described cases of duodenocolic fistula secondary to peptic ulceration in the second and third parts of the duodenum, while Ormandy and Bargen (1939) reported multiple fistulæ, including one between the colon and duodenum, occurring in a patient with ulcerative colitis.

The following two cases of duodenocolic fistula secondary to carcinoma of the colon are reported. The first patient was diagnosed at a stage when successful resection was possible, but the second, although detected before death, was too ill when first seen for any operative interference to be contemplated.

CASE REPORTS

Case 1.—Male, aged 74 years, admitted to the Medica Wards of the Royal Infirmary in June, 1947.

HISTORY.—In July, 1944, he had a hæmatemesis due to a duodenal ulcer, which was proved radiologically. He remained symptom-free on medical treatment until July, 1946, when he complained of passing red blood at stool; there was no diarrhœa. No cause for this was found other than small hæmorrhoids, and a barium enema was normal. He was seen again in September, 1946, when he stated that he had not had any further bleeding or ulcer symptoms; there was no bowel irregularity. Barium meal at this time showed a deformed duodenal cap but no ulcer crater. In May, 1947, he attended again, saying that he had remained perfectly well for seven months, but that in the last month he had been troubled by diarrhœa, loss of weight, anorexia, and listlessness. He described his stools as being watery, bulky, and pale—there was no blood or mucus and no offensive odour, but he thought that they floated. He was passing six to seven motions a day.

On admission he complained of colicky abdominal pain and an increase in the frequency of the diarrhœa.

ON EXAMINATION.—A tall elderly man with a high colour. No obvious wasting. Tongue coated but not atrophic, mucous membranes pale. Heart and lungs normal. Blood-pressure 120/75. No lymphatic gland enlargement. Abdomen showed some fullness and had a 'doughy' feel. No localized tenderness or mass. No free fluid. Liver and spleen were not enlarged. Rectal examination was negative. Central nervous system was normal.

Investigations.—Blood-count: Hb 61 per cent (8·6 g.); R.B.C. 3,600,000/c.mm.; W.B.C. 6800. A slightly macrocytic hypochromic anæmia was present (P.C.V. 3·5 per cent, M.C.V. 99c., M.C.Hb 24, M.C.HbC. 23·6 per cent). The film and differential count were normal. E.S.R. 34 mm. in 1 hour. Serum calcium 10·2 mg. per cent. Fractional test-meal: Hyperchlorhydria; no abnormal constituents. Stool examination: No occult blood, no pathogenic organisms, ova, or cysts. No undigested meat fibres. Examination for fæcal fats: 3-day excretion of fæces following 4 days' administration of 50 g. fat per day—the 72-hour specimen of fæces contained 11 g. of fat; total fat was 29 per cent of dry weight, split fat was 21 per cent of dry weight. (This indicated 93 per cent absorption of fat, which is within the limits of normality set by Cooke, Elkes, et al. (1946). These workers do not consider the percentage of split fat or the ratio of split fat to unsplit fat to be of importance.)

Radiological examination: Barium meal showed no evidence of ulcer or other abnormality. Follow-through showed a normal small-intestinal pattern. After 24 hours the meal was in the sigmoid. No evidence of neoplasm or fistula in small or large intestine.

Other investigations: Wassermann reaction negative. Glucose tolerance test: Fasting blood-sugar 68 mg. per cent; after 2 hours 115 mg. per cent. Urinary diastatic index, 7.

PROGRESS.—In view of the results of the investigations, and the clinical picture, it was felt that there was inadequate evidence of a neoplasm and that, in spite of the normal fæcal fat content, the case should be treated as one of non-tropical sprue. He was accordingly given a low-fat, high-protein, low-carbohydrate diet, and 10 mg. of folic acid twice daily for a week, followed by 5 mg. daily. After three days the frequency of his stools was reduced to once daily, and although still light in colour, they became normal in consistency. His Hb level rose to 69 per cent. His abdominal pain disappeared, but he did not gain weight. He was discharged home under his own doctor with instructions to attend for observation in the Out-Patient Department.

Two weeks later he was readmitted on account of recurrence of abdominal pain and diarrhœa. Examination of the abdomen revealed a vague mass in the right hypochondrium. Occult blood was present in his stools. The mass was considered to be in the region of the hepatic flexure and the diagnosis now changed to one of carcinoma of the colon.

A barium enema showed a filling defect in the region of the hepatic flexure with a fistulous communication with the duodenum (*Fig.* 291).

A surgical opinion was sought, and the patient was seen by one of us (E. P. H. D.). Operation was advised, and the patient agreed to this on condition that he be allowed to go home to settle his affairs. On Sept. 6 he was admitted to the Surgical Wards. He was given a high-protein diet, blood transfusions, and intravenous protein hydrolysate. Five days before operation, he was given a course of phthalyl-sulphathiazole.

AT OPERATION (E. P. H. D.).—On Sept. 15 (anæsthetic: pentothal, gas, oxygen and Tubarine). The abdomen was opened through a right paramedian incision. The growth was found to be in the hepatic flexure and was firmly fixed to the duodenum, but did not appear to have involved any other structure. The liver was free from secondary deposits but there were some enlarged glands in the colonic mesentery. The right colon was mobilized in order to see the extent of the duodenal involvement more clearly. This involvement was confined to a relatively small area on the anterior wall above the junction of the second and third parts. The growth was separated from the duodenum and the fistulous opening was seen to be approximately 0·5 cm. in diameter. The area of the duodenum concerned was widely excised, and then united by two layers of interrupted linen thread sutures in an end-to-end manner. The operation was completed by a right hemicolectomy with end-to-side union. The abdomen was closed with a stab drain in the flank.

Convalescence was uneventful and the patient was discharged on Oct. 4.

PATHOLOGICAL REPORT.—The specimen was that of a Grade 3 adenocarcinoma of the colon, which had involved the duodenum, causing a fistula. At the junction with the duodenum the cells had undergone squamous metaplasia. None of the glands examined showed involvement by growth.

POST-OPERATIVE PROGRESS.—The patient was seen in the 'follow-up' clinic on Feb. 11, 1948, and apart from a recent attack of 'flu' was well. He had put on weight and had normal bowel action.

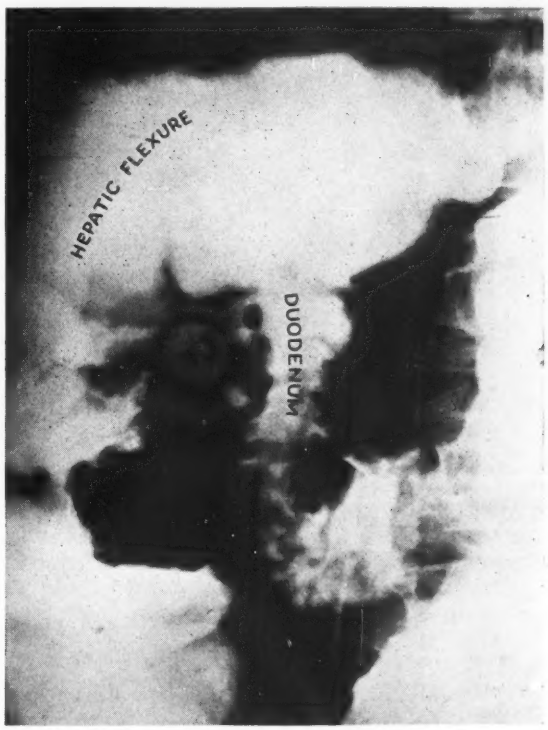

FIG. 291.—*Case* 1. Barium enema showing fistulous communication with duodenum.

On March 18 he was readmitted on account of progressive asthenia, but no further diarrhœa. On examination he was found to be anæmic, but his general nutrition was quite good. A hard nodular mass was found attached to the liver edge and the liver itself was enlarged. A blood-count showed Hb 44 per cent, R.B.C. 2,390,000. The cells were normocytic and hypochromic. A sternal puncture revealed normoblastic erythropoiesis with some maturation arrest of the normoblastic series.

The patient was given two pints of packed red cells and was discharged from hospital feeling much improved, but there can be no doubt that the mass in the abdomen represented a secondary deposit.

Case 2.—Male, aged 47 years, admitted to the Medical Wards of the Royal Infirmary in January, 1948.

HISTORY.—Cough and dyspnœa for many years, which caused him to leave his employment as a coal miner in 1943. In October, 1947, he developed diarrhœa with up to twenty-five yellow liquid stools daily, accompanied by lower abdominal pain and rapid loss of weight. Swelling of the hands, legs, and abdomen developed,

and on Dec. 17 he was admitted to his local hospital and thence transferred to the Royal Infirmary.

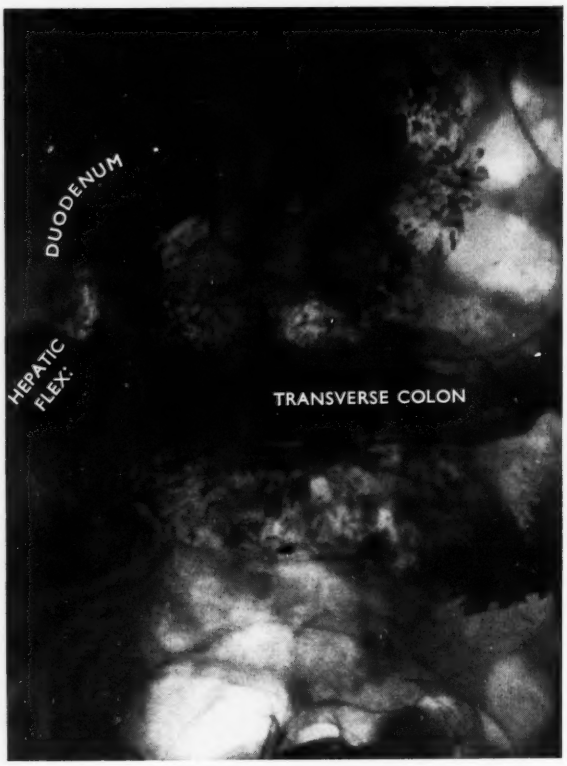

FIG. 292.—*Case* 2. Barium enema showing filling of the duodenum due to the fistula.

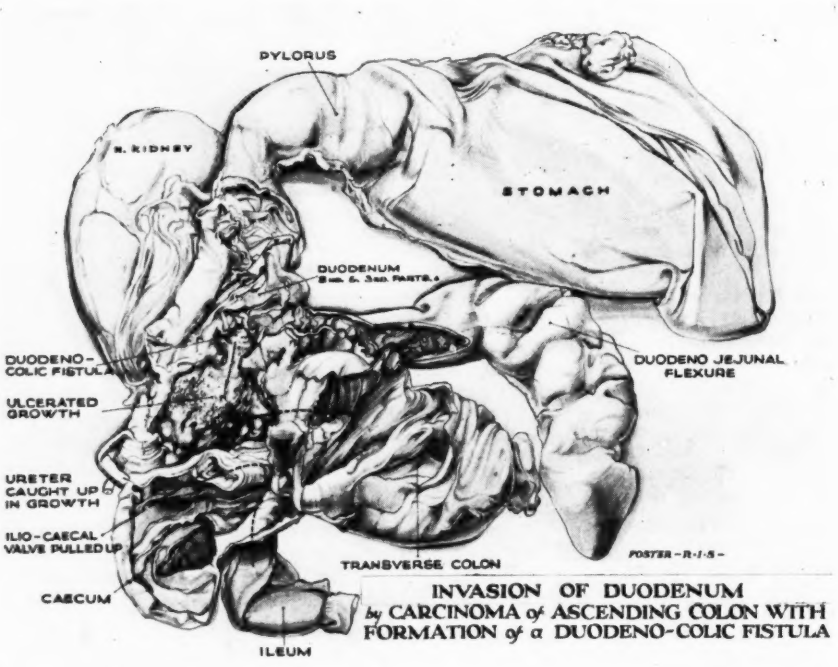

FIG. 294.—*Case* 2. Drawing of autopsy specimen.

ON EXAMINATION.—Extreme cachexia, no lymphatic gland enlargements. Tongue furred but not atrophic. Œdema of hands and legs. Heart normal. Lungs presented signs suggesting bilateral basal bronchiectasis. Abdomen distended, no mass or tenderness present, and

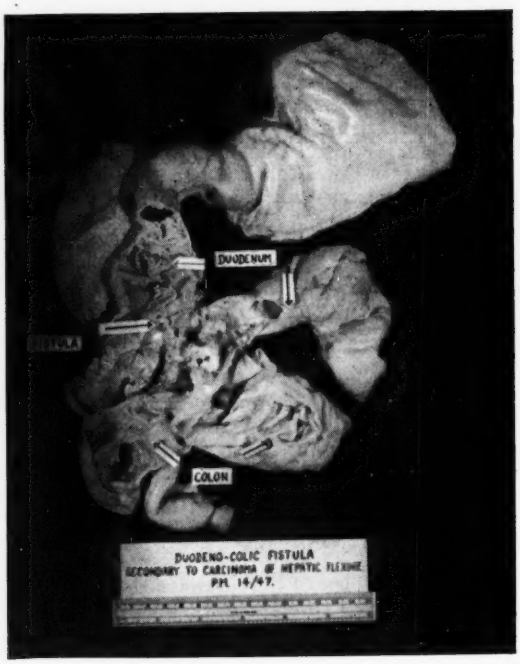

FIG. 293.—*Case* 2. Autopsy specimen.

no free fluid. Rectaex lamination negative. Central nervous system normal. Marked finger clubbing.

Investigations.—Blood-count: Hb 62 per cent, R.B.C. 3,290,000/c.mm., C.I. 0·94, W.B.C. 11,800, Polymorphs 71 per cent, Lymphocytes 23 per cent. The red cells were slightly macrocytic and hypochromic. Urinalysis: A trace of albumin. Nil else. No amyloid casts. Serum-Proteins: Total protein 4·7 per cent, albumin 3·2 per cent, globulin 1·6 per cent. Stools: No occult blood.

Radiological Examinations: Chest showed bilateral basal bronchiectasis. Barium enema revealed a filling defect in the region of the hepatic flexure with a fistula into the duodenum. Some barium had refluxed as far as the stomach (*Fig.* 292).

PROGRESS.—The patient was too ill for further investigations. His condition steadily deteriorated until death ensued.

AUTOPSY.—Both pleural sacs obliterated by adhesions. The lower lobes of both lungs showed advanced bronchiectasis with recent bronchopneumonia. There was no free fluid in the abdomen. The liver was small and fatty, and the spleen atrophied. A carcinoma was found to be present in the ascending colon and had infiltrated the lateral abdominal wall. The growth was adherent to the duodenum posteriorly and had formed a fistulous connexion with it, the opening of which was 1 in. in diameter. The right ureter was also involved in the

growth. (*Figs.* 293, 294.) A radiograph of the barium-filled specimen is shown in *Fig.* 295.

Microscopical Report.—In the colon the growth is on the whole fairly well differentiated with good acinus formation, but where it has invaded the wall of the duodenum differentiation is poor.

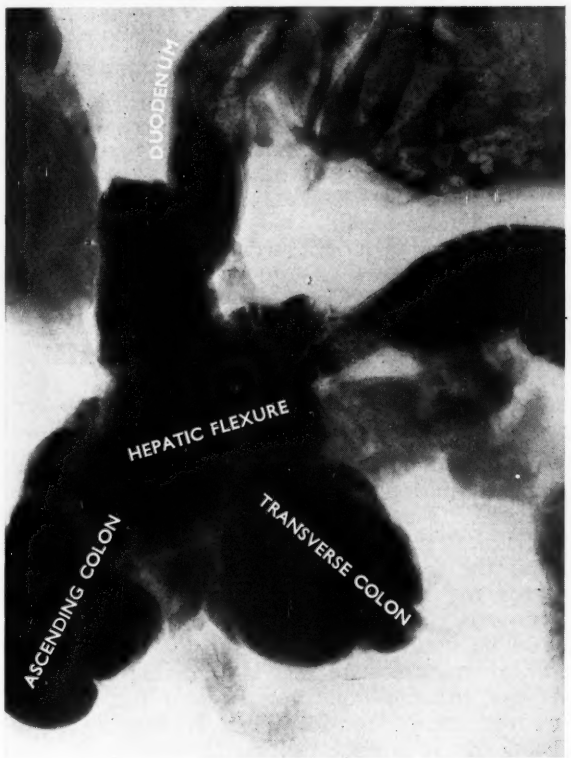

FIG. 295.—*Case* 2. Radiograph of autopsy specimen filled with barium.

DISCUSSION

The sudden onset of diarrhœa and rapid loss of weight, with gross nutritional disturbance, together with steatorrhœa and macrocytic anæmia, are all well recognized in cases with fistulous communications between the stomach or small bowel and the colon. Fairley and Kilner (1931) recorded three cases of gastrojejunocolic fistula which showed a macrocytic anæmia and steatorrhœa. Two of these cases had high gastric hydrochloric acid secretion and responded temporarily to liver extracts. It was suggested that malabsorption from the small intestine, resulting from the short-circuiting action of the fistula, was the factor responsible for deficiency of the anti-pernicious anæmia principle and the increased fat content of the stools. In *Case* 1, although the fæcal fat estimations were within the normal range (Cooke, Elkes, et al., 1946, and Black, Bound, and Fourman, 1947), it was considered that the syndrome was one of deficient intestinal absorption, due in all probability to a fistula. The history of diarrhœa, with pale bulky stools, loss of weight, and macrocytic anæmia, supported this view. But the absence of occult blood in the stools, the normal barium meal and follow-through examinations, and the initial response to folic acid and diet served to mislead one of us (J. F. G.) until a small mass became palpable in the abdomen and occult blood appeared in the stools. It is to be regretted that owing to technical difficulties at the time, no sternal marrow biopsy was obtained, although the presence or absence of a megaloblastic reaction would hardly have facilitated earlier diagnosis.

This case supports the findings of Mindline and Rosenheim (1935), Fossati (1938), McPeak (1940), and Feldman (1942), that the diagnosis of these cases presents considerable difficulties. All these observers stress the fact that gastro- or duodeno-colic fistulæ cannot be excluded by a barium meal and follow-through examination alone. It is the exception rather than the rule that this form of examination detects the lesion. It is only when the examination is made by a barium enema that the lesion is shown. This is probably accounted for by the positive pressure under which the examination is made, together with the propulsive power of the bowel in the evacuation of the barium. The value of air replacement must also be mentioned, for the full colon may overshadow the fistulous opening.

It is realized that the surgical procedure adopted in this case was not of a very radical nature. It was, however, not felt justified to subject a man of 74 years to any greater procedure. Linton (1944) described two cases where extensive resection was practised, and these are the only other cases we have found reported where resection has been successfully carried out. He described the cases of two males aged 59 and 45 years respectively. The operation was one of considerable magnitude and was conducted in two stages. The first stage consisted of an exclusion type partial gastrectomy, together with an ileotransverse colostomy with complete division of the transverse colon, the non-functioning part draining via the fistula. The fistula thus being short-circuited, the next three weeks or so were spent in improving the nutritional state of the patient. The gross malnutrition of these patients, and the difficulties of restoring nutrition to normal, form one of the greatest problems in the management of these cases. The second stage consisted in the removal of the antral part of the stomach, the duodenum, the head of the pancreas, and a right hemicolectomy, the bile-duct or gall-bladder being implanted into the jejunum. The pancreas was ligated and not anastomosed. The post-operative time of survival was not long, being 10 months in the first case and 8 months in the second.

Case 2 was unfortunately seen too late for any effective treatment to be carried out; he was not able to take the diet necessary for an accurate fæcal fat examination, and the differential diagnosis lay between amyloid disease secondary to long-standing bronchiectasis and gastrocolic fistula. As in *Case* 1, there was a tendency to macrocytosis of the red blood-cells. The sudden onset of diarrhœa with rapid wasting, associated with deficient intestinal absorption, occurring in a patient near middle age, with a short history of illness, should be regarded as due to some form of malignant fistulous communication between stomach or small bowel and colon, until proved otherwise.

SUMMARY

Two cases of duodenocolic fistula due to colonic carcinoma are described. In one, successful resection was carried out, but the other was seen too late for operation to be possible. The difficulties in diagnosis are pointed out, and the importance of a barium enema examination in the detection of such cases is stressed. The literature is briefly reviewed.

We wish to thank Mr. W. J. Lytle, Professor E. J. Wayne, and Dr. E. W. Skipper for kindly allowing us to publish these case notes, and Dr. L. C. D. Hermitte for the pathological reports.

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GLOSSOPHARYNGEAL NEURALGIA: EXTRACRANIAL NEURECTOMY

BY JOHN T. MORRISON, LIVERPOOL

SUCH excruciating pain as characterizes tic douloureux demands our utmost efforts to give relief. In the case of the commoner variety, that affecting the sensory branches of the 5th cranial nerve, surgical methods have now reached a certain degree of standardization. In that variety, however, in which the 9th cranial nerve is involved, no less painful but apparently much less common, there seems to be still considerable discussion. Theoretically, no doubt, permanent relief will only be given by intracranial preganglionic section, but relief up to ten years in one case and eight years in another is recorded in patients operated on by my colleague, Professor Herbert Williams, who avulsed the nerve by the extracranial approach.

As many of these patients are elderly and often are worn out by the pain they have suffered, the problem is a very real one, as to whether the intracranial operation, preferable on purely scientific grounds, is justifiable in practice. It would appear that in a number of instances the answer should be no. It then becomes important to consider carefully the technical aspects of the extracranial operation, at no time a simple or easy procedure owing to the depth of the jugular foramen from the surface and the anatomical complexity of the relations of the nerve as it emerges from the skull.

It might at first sight appear that local anæsthesia was ideal, but experience of the operation suggests that the necessity for prolonged retraction and the tilted position of the head renders local anæsthesia unsuitable. No doubt the addition of pentothal would help to overcome this objection. A non-toxic anæsthetic in the hands of a skilled anæsthetist is, however, probably preferable.

The number of those who have written on this subject is not large. Without exception they seem to have used an incision along the anterior border of the sternomastoid muscle. This is apt to be an unsightly incision, and suffers from the disadvantage that it is the upper extremity of the incision only which lies over the point we seek to reach. It is always preferable that an incision should be placed so that its midpoint lies over the area it is desired to expose. This objective is gained if the line of the classical neck incision is used, starting rather behind the mastoid process and curving forwards towards the hyoid bone, with a slight convexity downwards. The posterior two-thirds of this line should be used.

Most authors state that they divide at least in part the sternomastoid muscle, and this certainly improves access considerably. It has a further advantage. It at once exposes the spinal accessory nerve, and in the cases where this nerve crosses superficial to the internal jugular vein (at least 50 per cent), it is a most valuable guide in the deeper dissection. Farther forward the first useful landmark is the posterior belly of the digastric muscle, and it is recommended that this be clearly defined. It is then retracted upwards and forwards together with the external carotid artery lying deep to it. The stylohyoid muscle is not seen. The stylopharyngeus muscle passes forwards deep to the artery and its fibres can be seen but need not be further defined. The styloid process can be felt posteriorly, and when it is exceptionally long it might be helpful to crack it across near its base. In the case to be referred to it was quite short and gave no trouble. Traction at this stage is apt to be rather severe. It tilts the angle of the jaw forwards and upwards, and may exert such pressure on the lowest branch of the facial nerve as to cause some temporary paresis.

Returning now to the spinal accessory nerve, it is easy to trace this structure upwards and forwards as it crosses the internal jugular vein. As it goes, however, it begins to sink through a fascial layer, a thin but quite strong and well-defined fascial layer. Traced upwards this layer is found to be firmly attached to the outer edge of the jugular foramen. Presumably it is an upward extension of the carotid sheath. If the fascia is carefully slit up over the course of the nerve—this fascia

appears to be avascular—one is led directly to the jugular foramen at the point where the 9th, 10th, and 11th cranial nerves emerge in front of the jugular vein. The yellowish or reddish tint of the jugular ganglionic swelling on the vagus is quite striking. In order to display fully the nerves as they emerge, it is necessary to divide the small inferior petrosal sinus which comes out in front of the nerves and meanders downwards and backwards to join the internal jugular vein about 1 in. below the foramen.

The glossopharyngeal nerve is the foremost of the three, and is decidedly smaller than the other two. It turns away quite sharply forwards, crossing the internal carotid artery—here embedded in fascia, but easily palpable. There is a pharyngeal branch of the vagus, which also runs forwards, but this takes a more oblique course and lies at a decidedly lower level. The glossopharyngeal is seized with forceps, divided, and its proximal part avulsed by steady traction.

In a certain proportion of cases the spinal accessory nerve passes from the jugular foramen downwards and backwards deep to the internal jugular vein. One would then be compelled to divide the fascia over the vein and nerves without the assistance of the spinal accessory as a guide. The 9th and 10th, however, lie in such close relationship to the vein, that one cannot visualize the possibility of mistake, providing the vein is followed right up to the jugular foramen.

Some pains have been taken to describe this approach in detail, because some of the routes referred to in the literature appear unnecessarily severe, calling for division of the posterior belly of the digastric and the stylohyoid muscles, and even of the external carotid. In these the object is to pick up the glossopharyngeal as it lies below the lower border of the stylopharyngeus muscle. In my case the nerve was not at all easily visible in this situation, and in any case it must be followed right up to the foramen if one is to have the best chance of avulsing the ganglia on its root along with the nerve.

Avulsion of the nerve is followed by anæsthesia of the tonsil, fauces, and posterior part of the tongue. The patient is conscious of this loss of sensation, and it interferes to some extent with the swallowing reflex. The terrible paroxysms of pain cease of course immediately, but some residual discomfort may remain at least for a time in the distribution of one or other of the branches of the glossopharyngeal.

CASE REPORT

Miss R., aged 66. Quite suddenly, some three months previously, she felt a severe pain in the neighbourhood of the right ear. It came on without warning while she was at work in the house and was felt both in front of and behind the ear. It lasted some minutes and then passed off, but almost every day since, attacks have occurred and they tend to get more frequent and last longer. The pain was agonizing. She has noted that the attacks are often precipitated by opening her mouth widely and particularly by yawning. After a time the pain was felt also in the back of the tongue and the right side of the throat.

She is a stoutish, short-necked individual who looks her age. She was obviously worn out. The pain did not seem to be brought on by anything except the movement of the jaw referred to.

The operation already described was performed on Feb. 5, 1946, under general anæsthesia (Dr. Bennett Jones) without any upset to the patient. The following day quite a severe attack of pain in the neighbourhood of the ear was experienced, but she found already that she could open her mouth widely without bringing on the spasms of pain. There were no further severe attacks, but she did complain from day to day of slighter pains, both in front of and behind the ear. These are now becoming less frequent and are comparatively trivial. To relieve them she takes a couple of aspirin tablets occasionally.

She is immensely grateful for the abolition of the agonizing neuralgia, from which she used to suffer. There is still some difficulty with swallowing dry foodstuffs, but there is an improvement in this respect and it is easily overcome by taking a little drink of water. The right side of the back of the tongue, the right tonsil, fauces, and the right side of the soft palate are, of course, insensitive. The slight degree of facial paresis which followed the operation has disappeared.

COMMENT

It would appear that central stimulation of the area from which the nerve is torn, must have accounted for the attack of pain on the day after the operation. The same thing was noted by Professor Herbert Williams in the two cases he has allowed me to quote. I would suggest that the same explanation holds good for the slighter sensations of pain still experienced. Had they been due to a neuralgic condition in auricular branches of the glossopharyngeal nerve still left behind, it would be improbable that they would show the marked tendency to improvement which is present. At the operation, approximately 1 in. of the nerve was pulled out of the foramen. It is hard to see how this could be done without avulsing the jugular and petrous ganglia with it, as these ganglia lie, the one in the foramen, the other on the upper edge of the opening. This I believe is the explanation of the permanence of the results obtained by Herbert Williams. In other words, avulsion of the nerve performed in this way is in effect a preganglionic section.

In regard to the ear pain of which my patient still complains, it is just conceivable that this is due to stimulation of the auricular branch of the vagus which has connexions with the petrous ganglion of the glossopharyngeal nerve. This theory should perhaps receive consideration, although against it is the fact that the vagal branch appears to be distributed only behind the ear.

Removal of carious teeth at a later date finally disposed of the pain complained of.

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SHORT NOTES OF RARE OR OBSCURE CASES

HÆMANGIOMA OF THE SIGMOID COLON
REPORT OF A CASE

By C. C. HOLMAN

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JUDGING by the available literature, hæmangioma of the large bowel is a rare disease. Judd and Rankin (1922) quote Rokitansky, Virchow, and Kaufmann as having said that angiomas of the gastro-intestinal tract are extremely rare. Geschickter and Keasbey (1935) state that out of 570 hæmangiomas listed in the Johns Hopkins Hospital, only 10 occurred in kidney, spleen, intestine, or mesentery. McClure and Ellis (1930) collected 25 cases of hæmangiomata of the intestine, but in only one was the colon affected.

FIG. 296.—After barium meal, showing opacities in the left iliac fossa.

CASE REPORT

The patient, a girl aged 9, first came under observation in 1939, at the age of 15 months. She then had a large hæmangioma of the right hand and a smaller one of the tongue. The latter responded well to circumvallation with a diathermy needle. The hæmangioma of the hand was excised, as far as possible, but had to be treated on two subsequent occasions, by injection of sclerosing agents, before a cure was obtained. In 1942 she returned with a large hæmangioma in the suprascapular region. This was treated on similar lines to the hand, first by excision and later by injection of the residuum. In October, 1947, she was again admitted to Hospital with a history that, during the preceding six weeks, she had been passing considerable quantities of blood per rectum. Her hæmoglobin at the time of admission was only 54 per cent. Rectal examination and sigmoidoscopy failed to reveal the source of the bleeding. As it seemed possible that the bleeding might come from a Meckel's diverticulum, she was given a barium meal (*Fig.* 296). In his report Dr. Hollingsworth made the following observation: "There are multiple small scattered opacities in the region of the left iliac fossa. Their texture does not show the mottled characteristics of calcified glands, and a number of them show the features of phleboliths, which is somewhat abnormal at this early age. Is there any possibility of an angiomatous malformation, possibly involving the large bowel and giving rise to the rectal hæmorrhage?"

FIG. 297.—The hæmangioma as seen during the operation.

On the strength of this instructive report it was decided to operate, after preliminary blood transfusion and a course of sulphasuccidine.

AT OPERATION.—On opening the abdomen on Nov. 7 an extensive hæmangioma involving the upper part of the sigmoid at once presented. The condition is well shown in the excellent photograph (*Fig.* 297), taken by Mr. Pamely, of the Radiotherapy Department. The affected bowel was resected and immediate anastomosis performed. Recovery was uneventful.

THE SPECIMEN.—Dr. Bryan reported as follows:—

Macroscopical: "Colon unopened, 7·5 cm. long, 5·5 cm. in circumference. At one side of the mesentery there is an irregular collection of dark-red to blue-black smooth lobular swellings 4·5 × 1·5 cm. Protruding into the lumen there is a swelling 5·5 × 4·5 cm. The mucosa is stretched over the lobular surface of this tumour and there is a little shallow ulceration in the centre. On section the tumour is seen to be composed of irregular blood spaces, some of which contain calcified nodules."

Microscopical: "A cavernous hæmangioma. The majority of the blood spaces have relatively thick fibromuscular walls."

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A CASE OF LOCALIZED OSTEITIS FIBROSA OF THE SKULL

By MARGARET LESLIE and DAVID STENHOUSE
FROM THE GLASGOW AND WEST OF SCOTLAND NEUROSURGICAL UNIT

Comparatively few examples of osteitis fibrosa of the type localized to one lesion in the vault of the skull have so far been described. They occur in two forms, cystic and non-cystic, and cases of the former have been described mainly by German authors, including Wrede (1908), Frangenheim (1914), Kolbe (1918), and Petrow (1923). The non-cystic type is the rarer of the two, and Meyer (1930), in a search of the literature, quotes Frangenheim as having collected 6 cases, but since then we have been able to find no further reports. These cases present some difficulty in diagnosis and the following is brought forward as illustrating this and as indicating a satisfactory method of treatment.

Fig. 298.—Anteroposterior view of skull.

CASE REPORT

The patient, C. B., aged 22, an able-bodied seaman, was admitted to the Neurosurgical Unit, Killearn Hospital, under the care of Mr. Sloan Robertson on Aug. 28, 1945, complaining of a swelling on the left side of the head. This had been present and static for about two years, but the patient thought it had been increasing in size during the past two months. There had been no complaint of headaches, vomiting, or diminution of vision or hearing.

On Examination.—There was a large hard swelling palpable over the left parieto-occipital region. It measured approximately 7 × 9 cm. and was raised above the general contour of the skull by about 2·5 cm. at its greatest height. No other abnormality of the skull could be detected on percussion or auscultation, but the left side of the face appeared smaller than the right, especially above the level of the nose. General examination revealed a healthy young man with no symptoms referable to any system, and in particular no abnormality was detected in relation to the C.N.S.

X-ray examination showed a bony swelling in the left upper parietal region, measuring approximately 9 × 8 cm. (*Figs.* 298, 299). Its medial margin lay just to the right of the sagittal suture. The surface of the swelling was smooth and well defined. Clearly demarcated from the surrounding normal bone, it rose rather abruptly to a height of about 2 cm. above the neighbouring surface. Tangential views showed that the external table and diploë were mainly involved, and were indistinguishable from one another. The inner table was slightly depressed. Its inner surface was intact, but its outer surface was eroded into a series of ridges and hollows which presented themselves in the lateral view as a coarse granular appearance. X-ray pictures of the excised specimen show the changes more clearly (*Figs.* 300, 301).

Fig. 299.—Lateral view of skull.

With a tentative diagnosis of tumour, possibly sarcoma, ventriculography was carried out on Sept. 7 to exclude intracranial extension. The finding showed that the ventricles were in the normal position and of normal size. Owing to the appearance of middle-ear discharge on the left side, further exploration was delayed for over three months.

At Operation.—A bone-flap was turned and the altered area of bone removed. It was lifted with some difficulty, as it was rather adherent to the dura, but the adhesions were separated and a previously prepared Perspex plate, trimmed to the required size, was inserted, and the dura stitched up to the plate, which was then covered by pericranium. An uneventful convalescence followed this procedure.

THE SPECIMEN.—This consisted of an area of parietal bone measuring 9½ × 8½ cm. and covered by a thin fibrous tissue layer. In the centre the specimen was 2½ cm. thick, and this swelling tapered off in a very symmetrical manner to the entire periphery, where

FIG. 300.—Radiograph of excised bone, which has been bisected.

FIG. 301.—Radiograph of excised bone taken from inner aspect.

normal bone was reached. A saw cut bisecting the bone showed that the increase in thickness was due largely to an expansion of the diploic layer, which was only fairly well calcified and contained fibrous-like areas of varying size.

Very considerable congestion was present in the cut surface, which had a mottled dark-red colour (*Fig.* 302). Microscopical examination of decalcified portions taken throughout the specimen showed characters of a lesion of the osteitis fibrosa type (*Fig.* 303). The transition at the periphery from normal to altered bone was abrupt, and between the two there was an incomplete narrow zone of vessels containing hyaline thrombi. The bony trabeculæ then became altered in size and appearance, and assumed the characters of osteoid tissue, in which no mosaic patterns were visible. Between these areas the marrow spaces were entirely replaced by a very cellular fibrous tissue, the cells of which were of stellate type. Around some of the osteoid islets a few osteoblasts could be seen; these were outnumbered by small groups of osteoclasts, but the latter were nowhere numerous. The vascularity of the tissue varied throughout the sections, large dilated vessels being seen particularly under the outer table, and only a few small vessels showing at a deeper level. Both outer and inner tables were thinned out, and in the former a few small defects bridged by fibrous tissue were noted. The lesion was at the stage in which absorption of bone was in excess of formation. No evidence of xanthoma cells was noted.

Discussion.—The patient was sent to the Neurosurgical Unit as a possible case of malignant tumour of the skull. On clinical and radiological grounds the main diagnostic problem was to decide whether the bony swelling was a primary disease of bone or was secondary to an intracranial tumour. The possibility of meningioma was considered unlikely from the X-ray appearances, as the density of the involved portion of bone was rather less than normal and the changes affected the diploë mainly. It was also thought that a malignant lesion could be excluded by the absence of neurological signs, by normal ventriculography, and by the normal appearance of the underlying dura at operation. Even after removal of the abnormal bone a diagnosis was not made from the naked-eye appearances alone. After histological sections had been examined, the long bones and the pelvis were X-rayed, and normal bone structure found. Blood-chemistry examination, which was confined to estimation of serum-calcium (10·65 mg. per 100 ml.) and serum-phosphatase (alkaline 2·4 K and A units, acid 1·7 K and A units

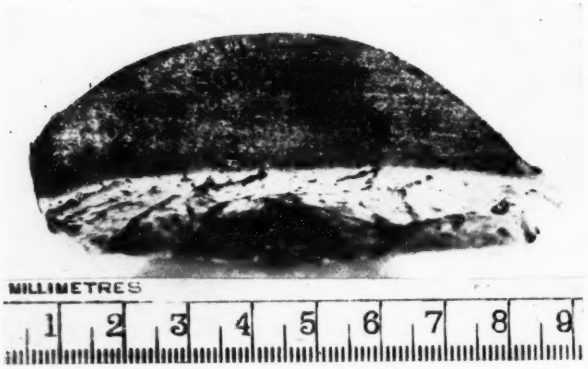

FIG. 302.—Showing cut surface of excised bone. (× ⅔.)

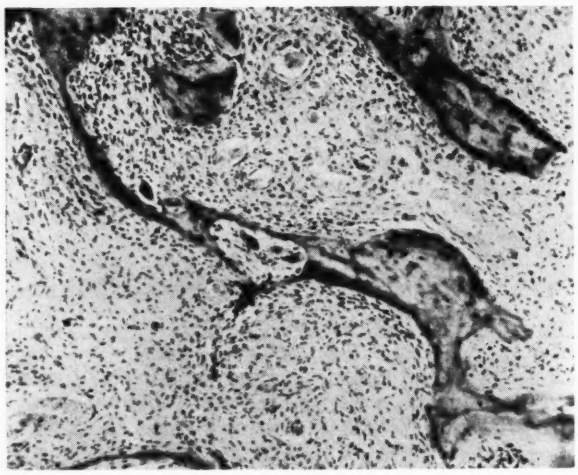

FIG. 303.—Section through central part of specimen. (× 85.)

per 100 ml.), showed figures within the normal range. It was only then that a diagnosis of osteitis fibrosa localisata could be made with some confidence.

Our thanks are due to Mr. Sloan Robertson and to the Medical Superintendent for permission to report the case.

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GROSS HYPERTROPHY OF THE PALMARIS LONGUS MUSCLE SIMULATING A TUMOUR OF THE FOREARM

By ROY GOULDING, London

CASE REPORT

History.—Leslie L., aged 22 years, an engineer by trade, first noticed a swelling on the volar aspect of the right forearm five years ago. The onset was fairly sudden and unrelated to any injury or accident. Rest and support by means of a sling and plaster for nine weeks brought about no change. In February, 1944, an exploratory operation was carried out at the County

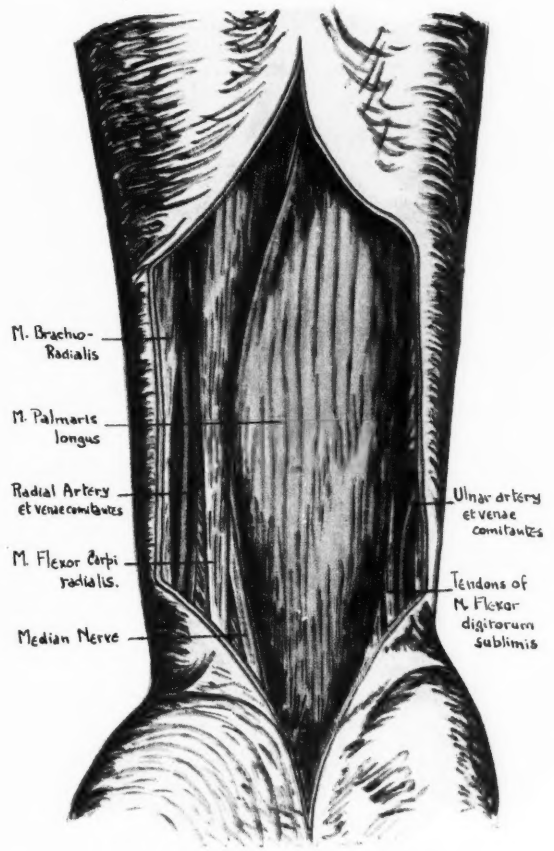

Fig. 304.—Sketch showing the position and form of the hypertrophied palmaris longus as seen on dissection.

Hospital, Pembury, and the report made reference to a "marked hypertrophy of the palmaris longus". Meanwhile the patient continued at work with the swelling still present. There was no alteration in the size, and only over the last twelve months was there any complaint of pain, then described as a "dull ache". The only other feature noted was a lump in the axilla that "came and went". General health was satisfactory, there was no cough, appetite was good, bowels were regular, and micturition was normal. Neither in the family history nor in the previous diseases was there anything relevant.

On Examination.—The patient was a well-built, fit-looking individual. Nothing abnormal was discovered in any of the principal systems and on radiography the lung fields were shown to be clear. On the volar aspect of the right forearm, rather towards the ulnar border, was an oval swelling about 2½ in. by 1½ in. immediately proximal to the skin creases and with its long axis in line with the arm. Its consistency was firm. The surface appeared to be lobulated and the margin was not well defined. There was no attachment to the covering skin, which was unaltered. On palpation no crepitation was detected, nor could the swelling be transmitted beneath the flexor retinaculum to the palm. There was some attachment to deep structures. Transillumination was not demonstrable and fluctuation could be elicited only in a transverse plane. Movements at the wrist and of the fingers were full and painless. Power was equal to that in the other arm. On flexion of the wrist the swelling became less prominent, though rather firmer. The lymph-nodes in both axillæ were slightly enlarged.

Temperature, pulse, blood-sedimentation rate, and blood-picture were all normal.

In the absence of cystic characters the diagnosis of the tumour seemed to lie between a subfascial lipoma, some type of myxoma or myxochondroma of the tendon-sheath, and an unusually placed muscle.

At Operation (Feb. 17, 1948).—Longitudinal incision revealed a grossly hypertrophied palmaris longus (*Fig.* 304). The muscle belly reached almost to the insertion. Precisely how far towards the common flexor origin the contractile tissue extended was not determined. It was decided that the greater part of the muscle should be excised. The extremely short tendon of insertion was therefore severed from its connexions with the palmar aponeurosis, the retinaculum, and the short muscles of the thumb. The muscle belly was then cut across about 4 in. proximally. At this level there was a noticeable tapering, but no tendon of origin was defined. The wound was closed, the skin sutured, and recovery proceeded uneventfully.

DISCUSSION

Thane (1882) refers to the palmaris longus as the most variable muscle in the body. Wanting in about 10 per cent of subjects, it exhibits many variations of form when it is present. The fleshy fibres may occupy the middle of the muscle, which then commences and ends by an elongated tendon, or the muscular structure may be placed towards the lower end, the upper part being tendinous. The whole muscle may be reduced to a mere tendinous band. A digastric muscle has also been observed. It is sometimes represented by a slip from the flexor carpi ulnaris or flexor digitorum sublimis. Occasionally there may be two long palmar muscles. With regard to the insertion there is a similar lack of uniformity.

One would expect to learn more about the palmaris longus and its remarkable variations from comparative anatomy; yet Cuvier (1805), in his classical treatise, fails to mention this muscle. A full account of its occurrence and form in different species is included in the work of Meckel (1829), but it is to Testut (1884) that one turns for a most exhaustive survey. He not only enumerates the many variations observed in man, but also describes in great detail the diverse arrangements found among the animal world. He explains these by the conception of a 'pronato-flexor mass' arising as

one from the medial epicondyle of the humerus and dividing below into a number of parts, the degree of development so attained being in accordance with the functions that the forearm is called upon to perform. At the one extreme are the reptiles, their limbs executing no more than simple, gross movements and the musculature being comparatively undifferentiated. At the other end of the scale are the higher vertebrates, masters of the most elaborate movement patterns and provided wth a correspondingly intricate muscular equipment. The numerous variations encountered among human subjects are then interpreted as stages, atavistic or progressive, within this wide range.

Wood Jones (1946), for his part, regards the palmaris longus as a muscle passing into phylogenetic decrepitude. Originally the most superficial of the three layers of the long flexors of the fingers, it has now lost its functional importance in respect of the metacarpophalangeal joints and its degenerated tendons are represented by the fascial sheet going by the term of palmar aponeurosis. Attention is drawn to these remnants in the pathological condition associated with the name of Dupuytren.

With this patient the question arises whether there is a similar formation in the other arm. No prominent swelling presents. Yet on clinical scrutiny there is a definite fullness above the wrist and an unusual resilience to palpation of the volar aspect. It will be interesting to observe whether any difference in power of flexion at the wrist exists after recovery from the operation.

I should like to record my grateful acknowledgement to Mr. H. J. B. Atkins for his valuable criticism and his permission to publish this article. My thanks are also due to Mr. C. P. Nicholas for his very helpful advice.

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PERFORATED JEJUNAL DIVERTICULUM

REPORT OF A CASE, WITH SOME COMMENTS ON THE AETIOLOGY

BY W. B. WATERFALL, PLYMOUTH

JEJUNAL diverticula are not commonly seen as a cause of symptoms. Butler, in 1933, published a paper based on 7 personal cases and the 13 which he was then able to find described in sufficient detail.

Fraser (1933), in describing a new case of what he aptly described as multiple false diverticula of the jejuno-ileum, referred to Case's series of 70 cases as the largest yet published, but thought that many of these should have been classified in one of the other sub-divisions of diverticula of the small intestine. Milnes Walker, in 1945, stated that the condition had been recorded on approximately 225 occasions, but that only 19 cases of acute inflammation had been reported prior to the 3 further cases which he then described. The case described here therefore appears worth recording.

CASE REPORT

HISTORY.—A farm labourer, aged 66, was admitted as an emergency on the evening of May 14, 1947. He had been quite well until two days before admission, when he complained of pain on the left side of the abdomen. It was aching in character, non-radiating, and associated with nausea but not with vomiting.

His bowels had not been open for the same period ; they were usually regular with the aid of liquid paraffin. There was no history of blood or slime in the motions, no urinary symptoms, no cough, and no loss of weight.

His appetite was usually good ; he had marked flatulence after meals but no pain, and therefore had never taken any medicine.

He had had a hæmatemesis six years previously. A barium meal investigation at that time had shown no abnormality, and he had made a good recovery with medical treatment.

ON ADMISSION.—Temperature 99.5° ; pulse 72 ; respiration 18. Tongue dry and furred. Not shocked. Heart and lungs clear. Abdomen tender on left, not distended. Muscle guarding fairly pronounced, but little true rigidity. Both the tenderness on palpation and the subjective symptom of pain were more marked over the lower left abdomen than the upper. Bowel sounds could be heard with the stethoscope.

P.R. some rather indefinite tenderness. No mass felt. Normal fæces on examining finger.

Provisional Diagnosis.—Acute diverticulitis of the descending or pelvic colon.

TREATMENT.—Expectant. Fowler's position. Enema given. Fluids only. Two-hourly pulse and temperature chart. Sedatives allowed.

PROGRESS.—On May 15 temperature rose, reaching 101° at 6 a.m. and 102° at 10 a.m. Pulse also rose, but less markedly, reaching 96 at 10 a.m. Re-examination now showed marked rigidity of the left abdomen, and bowel sounds could no longer be heard.

OPERATION (May 15, by W. B. W.).—Gas, oxygen, ether anæsthesia by Dr. Clements.

Left lower paramedian incision. Some dirty free fluid in pelvis, but no diverticulitis of pelvic or iliac colons. The appendix was not evidently inflamed.

On inspection of the transverse colon, a mass was found deep to the mesocolon. The incision was extended upwards, and this proved to be a mass of inflammation surrounding and arising from diverticula on the mesenteric border of the first loop of the jejunum, and to be surrounded by free pus. (*Fig.* 305.)

Resection was performed, not without anxiety for the safety of the main branches of the mesenteric vessels in the œdematous tissue, and the ends of the intestine were embedded and a side-to-side anastomosis was performed.

Penicillin-sulphathiazole powder was dusted around the anastomosis and the omentum drawn over it.

A tube drain was brought out through a stab at the outer border of the rectus, and the abdomen closed by layers.

POST-OPERATIVE PROGRESS.—The patient stood the operation well and made a surprisingly smooth recovery. Intravenous glucose-saline was continued for 60 hours, at the rate of 5 pints per 24 hours; sulphadiazine was administered, at first through the intravenous drip, at the rate of 5 to 6 g. per day to a total of 32 g.; and intravenous and later intramuscular penicillin was given in a dosage of 30,000 units 3-hourly to a total of 1,350,000 units. Feeding was commenced 24 hours after operation with small hourly sips of water, and was gradually increased; no vomiting occurred at any time.

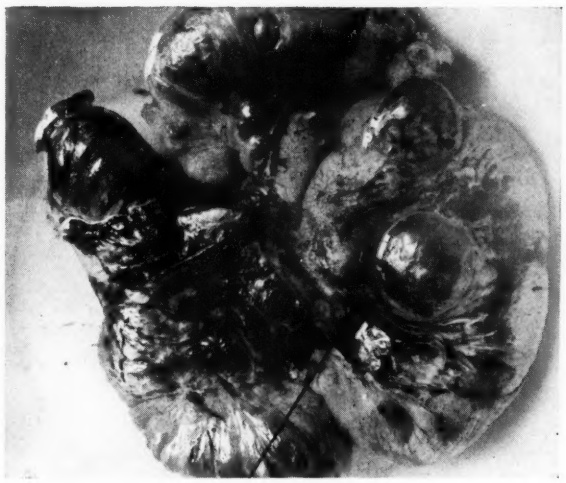

FIG. 305.—The excised intestine showing the perforation and the collapsed diverticulum behind it.

The drainage tube was shortened at 48 hours, and was removed on the fourth day. The abdominal wound healed cleanly, and the stitches were removed on the tenth day. He was encouraged to move freely in bed, but did not get up until the twelfth day. He was discharged seventeen days after operation, and has been in good health since.

Radiographs taken 4½ and 6 hours after a barium meal on Jan. 13, 1948, show no further diverticula, and no obstruction at the anastomosis.

DISCUSSION

It would seem probable, from the relatively mild symptoms and physical signs on admission, and especially from the fact that peristaltic sounds were then audible, that he had been suffering from an acute unperforated diverticulitis, that the perforation occurred as a result of the jolting of the ambulance journey to hospital, and that the peritonitis spread during the night. The left-sided symptoms would be explained by the watershed effect of the root of the mesentery.

The relation of the diverticula to the hæmatemesis of six years previously seems doubtful, but it is interesting that a radiograph taken with the object of demonstrating a possible gastric ulcer showed no abnormality.

The photograph (*Fig.* 305) shows the perforation and the collapsed diverticulum behind it. It also shows the position of the diverticula to be lateral to the mesenteric border, as stressed by Fraser (1933), and also the thin-walled nature as described by both Fraser and Butler (1933).

This patient was of the age and sex in which this condition has usually been recorded, and his previous freedom from symptoms, other than flatulence, was in keeping with the observations of the above authors. But it is noticeable that there has been no clinical evidence of the raised intra-alimentary pressure noted by Fraser as a major aetiological factor. This seems to support Butler's suggestion that traction of an arteriosclerotic mesenteric arteriole may be a major factor, since he has the degree of arteriosclerosis commonly present at his age, and since the diverticula may be seen to rise in positions corresponding to the laterally-placed passage of the vasa recta through the muscle wall.

I am grateful to Mr. C. M. Kennedy for allowing me to treat and to describe this case, and to Dr. Craig Mooney for radiological investigations and for the clinical photograph.

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CARCINOID TUMOUR OF THE SMALL INTESTINE WITH SECONDARY DEPOSITS IN VARIOUS ORGANS

BY PHILIP WRIGHTSON, DARLINGTON

A CASE is reported of carcinoid tumour of the small intestine with secondary deposits in lymph-nodes, liver, and heart muscle.

CASE REPORT

E. B., a man of 50 years, had complained for eighteen months of persistent tenesmus and diarrhœa with blood and mucus in the stools.

ON EXAMINATION.—There were clinical signs of hypertensive heart failure of moderate degree. The right lobe of the liver was felt two fingerbreadths below the costal margin and was hard. No ascites was detected. In the midline of the anus posteriorly there was a deep indurated ulcer. Above this there was some thickening of the posterior rectal wall, and on pressing this pus exuded from the base of the ulcer. The inguinal lymph-nodes were not enlarged.

Microscopical examination of the stools showed blood and pus; no amœbæ or cysts were seen.

Sigmoidoscopy revealed a diffuse proctitis and colitis; no neoplasm was seen.

Biopsy of the anal ulcer showed chronic inflammatory tissue only.

The colon filled well with a *barium enema* and there was no evidence of neoplasm.

PROGRESS.—The patient's general condition did not permit more than symptomatic treatment. He deteriorated steadily until his death four months later from heart failure. There was no sign of intestinal obstruction and no further enlargement of the liver in this time.

AT AUTOPSY.—

Peritoneal Cavity: There was no free fluid. The terminal ileum was acutely kinked about 7 cm. from the ileocæcal valve, and the mesentery was shortened in this region. At the apex of the kink a firm globular tumour about 3·5 cm. in diameter was felt through the bowel wall. The peritoneal surface over the swelling was normal. The ileocæcal lymph-node immediately related to the tumour was enlarged to 2·5 cm. in length and there was a chain of smaller lymph-nodes extending along the superior mesenteric vessels. The liver was moderately enlarged and studded with deposits.

On opening the ileum a globular tumour was seen projecting into the lumen from the mesenteric half of the circumference (*Figs.* 306, 307). It was covered with thin mucosa except at the summit where there was a small ulcer. It was partly obstructing the gut, so that the bowel above was a little dilated and the walls hypertrophied. On section the tumour was golden yellow in colour. The muscle of the intestinal wall underlying the tumour was thickened and broken up into fasciculi by the growth; about 3 cm. of the bowel wall was involved. The rest of the small and large intestines was free from growth. The walls of the descending colon and sigmoid were thickened and showed chronic inflammatory changes. There was a post-rectal abscess with massive surrounding fibrosis, communicating with the anal ulcer.

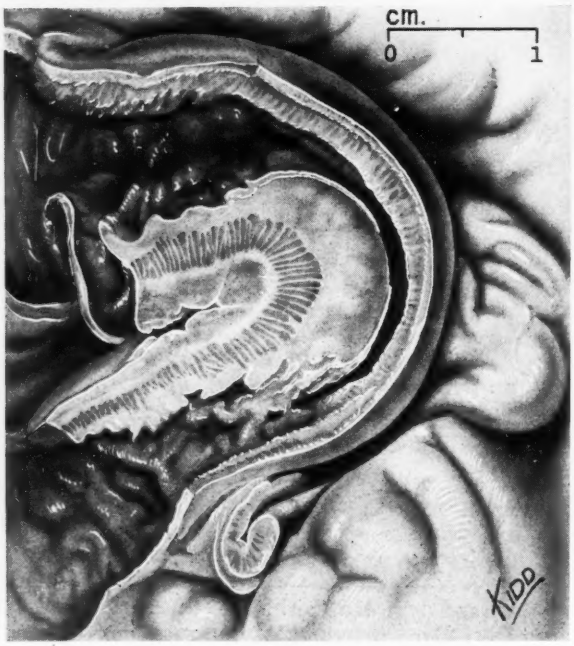

FIG. 306.—Primary carcinoid tumour of ileum, sectioned.

Many deposits of a golden yellow colour were present throughout the liver, varying in diameter from 0·5 cm. to 7 cm. (*Fig.* 308). The cut surface of the mesenteric lymph-nodes was similar.

Thorax: The lungs were congested; they contained no obvious metastases. The ventricular muscle of the heart was hypertrophied and showed marked brown atrophy. In the wall of the left ventricle there was a spherical deposit of the same yellow colour, 1·2 cm. in diameter, lying under the epicardium (*Fig.* 309). There were the usual changes of hypertensive heart failure.

Other thoracic and abdominal viscera were apparently free from metastasis.

Histological Report (Dr. H. A. Cookson).—"Carcinoid tumour of the small intestine of typical macroscopical and microscopical appearance (*Fig.* 310). Secondary deposits of similar structure in liver and heart muscle. Section from post-rectal tissues: chronic non-tuberculous abscess."

COMMENT

Oberndorfer (1907) first gave the name 'carcinoid' to this tumour of the small intestine.

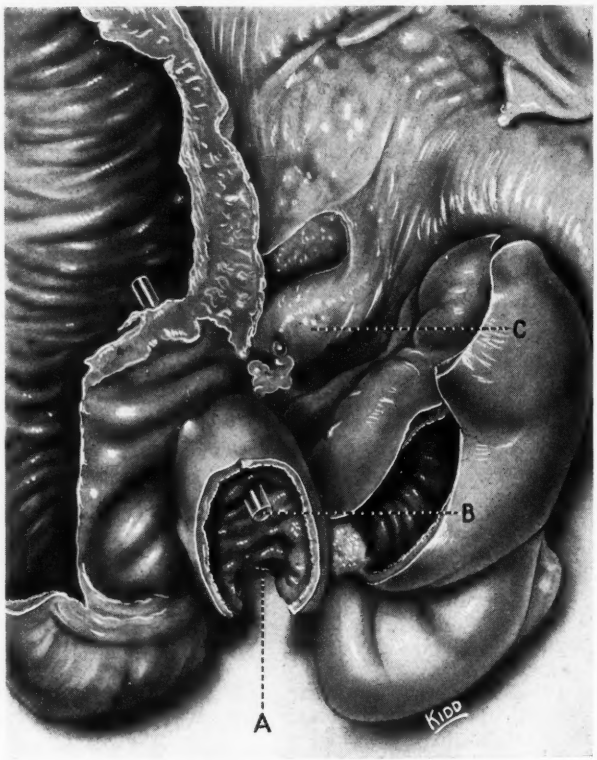

FIG. 307.—Terminal ileum and cæcum. A, Cut surface of tumour at right angles to picture plane; B, Glass rod through ileocæcal valve; C, Invaded node and contracted mesentery.

He stated that in his cases the growths were small and multiple, did not invade surrounding tissues, and did not metastasize; that they grew slowly, never reached a great size, and appeared to be harmless. Cases subsequently reported have modified this conception; it has been shown that the tumour is more often single than multiple, that it is of a size comparable with the adenocarcinoma of the small intestine, and that metastasis occurs in lymph-nodes, liver, and other organs. Cooke (1931), reviewing the literature, found 104 cases of carcinoid of the small intestine. Of these, 21 were considered to be malignant, 3 showing extension to the serosa in the region of the growth, 10 to the lymph-nodes, and 8 to the liver and other structures. All these malignant tumours were in the ileum. Raiford (1933) described a carcinoid of the sigmoid colon with abdominal metastases and a small secondary deposit in the lung.

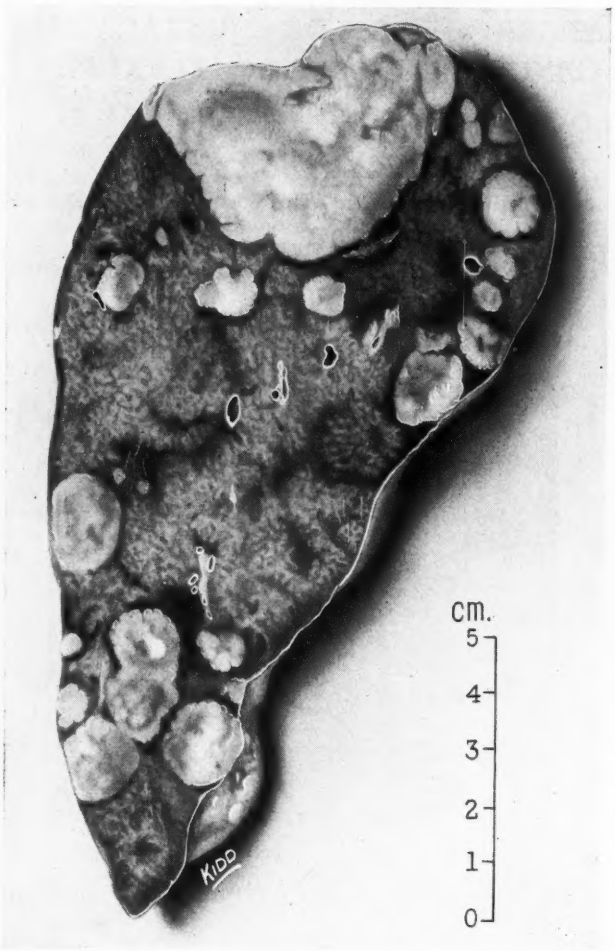

FIG. 308.—Secondary deposits in liver.

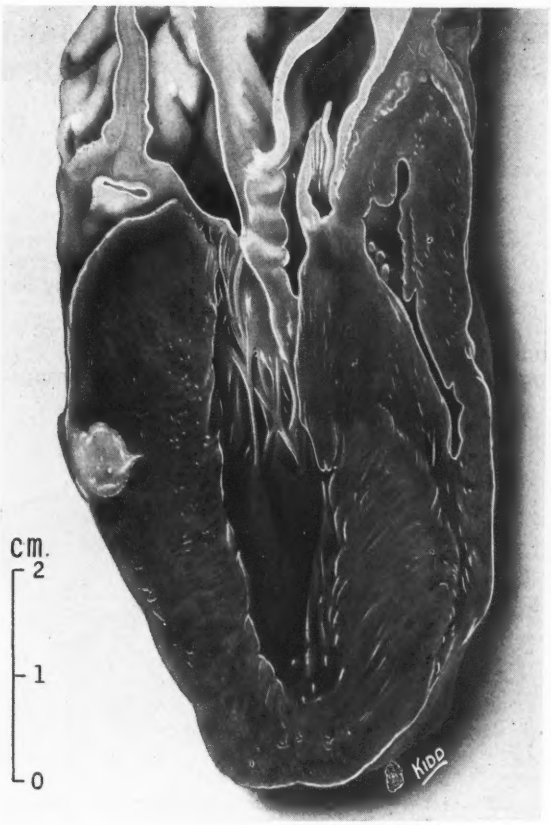

FIG. 309.—Secondary deposit in heart.

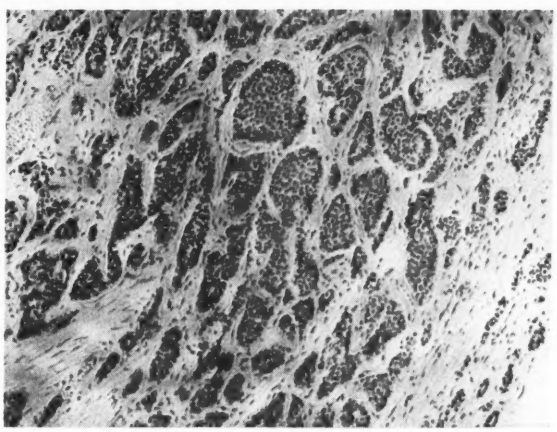

FIG. 310.—Photomicrograph of section of growth (low power).

In the case here reported metastases involved lymph-nodes, liver, and heart muscle.

Diagnosis.—Diagnosis is usually made at autopsy or at operation for obstruction. In Cooke's series, of the 21 cases with metastases 10 developed intestinal obstruction and 2 complained of wasting and anæmia only; the others were diagnosed at autopsy. Of the 83 benign growths, 75 were discovered at autopsy and 6 developed intestinal obstruction.

The case described here presented the problem of an enlarged liver, possibly due to secondary deposits from a primary growth which could not be detected. In such a case the good condition of the patient in the presence of extensive metastases might suggest a carcinoid tumour as the primary condition.

I wish to thank the Medical Superintendent of Cherry Knowle Emergency Hospital for permission to publish this case; Dr. H. A. Cookson, lately Director, Pathological Department, Royal Infirmary, Sunderland, for the histological report and for his encouragement and help; Dr. J. C. Wilson, the present Director, for the photomicrograph; and Professor F. H. Bentley for much valuable advice.

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EXCISION OF SECOND PRIMARY CANCER OF COLON (JUXTACÆCAL) 20 YEARS AFTER REMOVAL OF CANCER OF SPLENIC FLEXURE

By A. WILFRID ADAMS

CASE REPORT

The patient, C. H., 52 years of age, came first to me in November, 1927, with a two-years' history of dyspepsia and central abdominal pain. Bowel action was regular. He had lost 18 lb. in weight. There was a strong family history of carcinoma.

He had been seen by a physician the previous April and after radiography was diagnosed as a case of chronic appendicitis, but he refused operation. Constipation

FIG. 311.—Specimen of intussusception of transverse colon due to carcinoma removed in 1927 from C. H., male, aged 32 years.

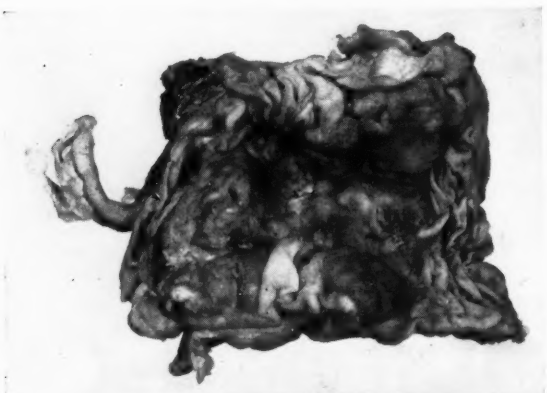


FIG. 313.—Specimen of juxtacæcal cancer coli removed in 1947 from C. H., male, aged 52 years.

developed and on Nov. 11, sudden severe abdominal pain and vomiting set in. He was admitted to the Bristol Royal Infirmary on Nov. 21.

Examination revealed a distended abdomen due to colonic obstruction by a mobile lump below the splenic region. Cæcostomy was performed on Nov 27, followed a fortnight later by radical removal of a large colonic carcinoma which had caused an intussusception of the splenic flexure (*Fig.* 311). Numerous paracolic glands were included in the excision and immediate re-union of the colon done. Recovery was uneventful and he worked until Autumn, 1947.

He returned on Oct. 22, 1947, with a history of three months' intermittent right abdominal pain. Borborygmi troubled him notably, but the bowels were regular save for occasional laxity. There was no loss of appetite or weight. He complained also of general weakness, occasional headaches, and insomnia.

He looked poorly, very pale and anæmic. Per abdomen and rectum the sole abnormality was a small

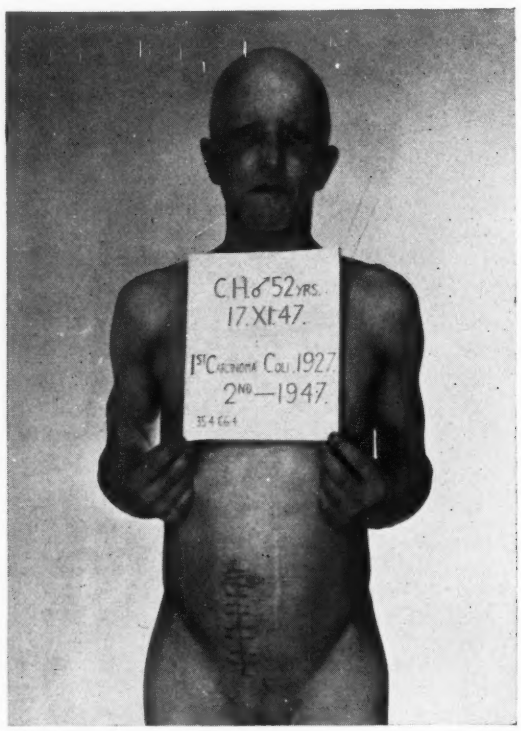


FIG. 312.—Photograph of abdominal scars after removal of two independent carcinomata of colon from C. H., in 1927 and 1947.

nodular mobile mass above the old cæcostomy scar in the right iliac fossa. Radiography with a barium enema showed the old anastomosis to be working well. The ascending colon was narrowed with a constant irregular outline at the site of the palpable tumour. Blood count: R.B.C. 3,700,000, Hb 42 per cent, W.B.C. 8,000. Blood-pressure 120/65. He was rested for a week and given two pints of whole blood and two pints of packed cells transfusion so that by Oct. 29 the Hb had risen to 84 per cent.

On Oct. 31, 1947, I resected a juxtacæcal colonic carcinoma and performed end-to-side union of ileum with descending colon. No secondaries were found in the abdomen. On Nov. 17 the patient (*Fig.* 312) was up and progressing well; the bowel was acting well.

He has since been examined (Aug. 30, 1948) and is still free from any signs of recurrence. He is a busy factory worker, apparently perfectly fit.

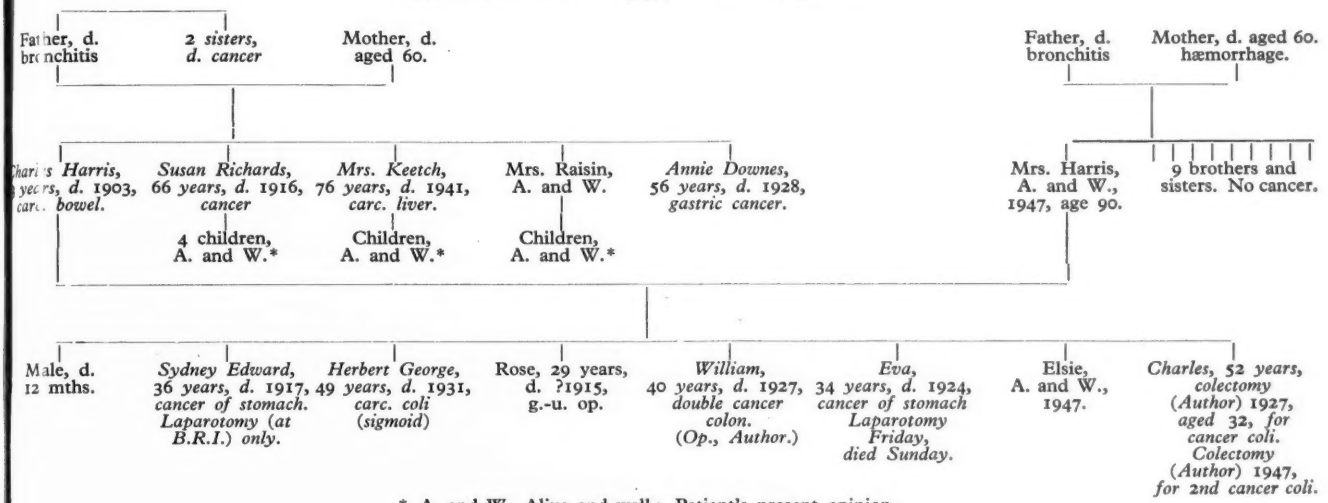
DISCUSSION

This records an apparent cure of a florid cancer of the left colon in a young man in whom, twenty years later, a new primary scirrhous cancer coli (juxtacæcal) has been removed (*Fig.* 313).

To this rare personal history of the disease is added an exceptional story of familial cancer. The facts, up to date, are :—

patient has afforded an exceptionally good example of protracted cure of a colonic cancer (20 years) by surgery. His recent tumour was obviously an

FAMILY HISTORY TO 1947, OF C. H., 52 YEARS


Father, d. bronchitis
2 sisters, d. cancer
Mother, d. aged 60.
Father, d. bronchitis
Mother, d. aged 60. hæmorrhage.
Charles Harris, years, d. 1903, carc. bowel.
Susan Richards, 66 years, d. 1916, cancer
4 children, A. and W.*
Mrs. Keetch, 76 years, d. 1941, carc. liver.
Children, A. and W.*
Mrs. Raisin, A. and W.
Children, A. and W.*
Annie Downes, 56 years, d. 1928, gastric cancer.
Mrs. Harris, A. and W., 1947, age 90.
9 brothers and sisters. No cancer.
Male, d. 12 mths.
Sydney Edward, 36 years, d. 1917, cancer of stomach. Laparotomy (at B.R.I.) only.
Herbert George, 49 years, d. 1931, carc. coli (sigmoid)
Rose, 29 years, d. ?1915, g.-u. op.
William, 40 years, d. 1927, double cancer colon. (Op., Author.)
Eva, 34 years, d. 1924, cancer of stomach Laparotomy Friday, died Sunday.
Elsie, A. and W., 1947.
Charles, 52 years, colectomy (Author) 1927, aged 32, for cancer coli. Colectomy (Author) 1947, for 2nd cancer coli.

* A. and W., Alive and well ; Patient's present opinion.

1. That a cancerous disposition may be inherited is strikingly proven in this patient's family history.
2. The pre-disposition to cancer confined to a single system (alimentary) is shown.
3. Despite the awful familial susceptibility, my independent second primary growth with no unusual histological characteristics.

This story is carefully reported for its potential value in the study of the aetiology of this great enemy of mankind.

SIMULTANEOUS INJURY OF RECTUM AND URETER

BY KENNETH G. F. MACKENZIE

NORTH STAFFS ROYAL INFIRMARY

AN unusual case is reported of perforating injury of both rectum and ureter by the same agent. This and the strange method of its production render it worthy of record.

CASE REPORT

A boy, aged 14 years, was admitted to hospital as an emergency on June 28, 1947. A few hours before, he had been climbing a pole supporting the roof of a building, encouraged from below by a friend with a billiard cue. He fell from the pole on to the billiard cue and immediately experienced severe abdominal pain, which had since become more intense and given rise to vomiting.

ON EXAMINATION.—The boy was shocked, with a pulse of 120 and of poor volume. He was extremely restless and difficult to examine on account of the obvious severity of his pain. There was no sign of any external injury. The abdomen showed a generalized tenderness and rigidity without any localization. Rectal examination was very painful ; no perforation could be felt and there was no blood on the examining finger.

The pre-operative diagnosis was a perforating injury of the rectum caused by a billiard cue entering the anus. Resuscitative measures were instituted, a plasma drip started, and laparotomy undertaken six hours after injury.

AT OPERATION.—A right paramedian incision was made, excising an old appendicectomy scar. A small clean puncture of the anterior rectal wall was seen at the level of the peritoneal reflection, and this was sutured in two layers. Further examination showed a laceration of the posterior parietal peritoneum just above the pelvic brim on the left side, and from this urine was escaping.

A track ran retroperitoneally in the direction of the kidney, and it was obvious that either the ureter or renal pelvis had been opened. Further exploration was not considered advisable, so one retroperitoneal and another intraperitoneal drain were brought out through a stab wound in the loin, the peritoneal tear being sutured. A rectal tube was passed well above the level of the bowel injury. Sulphonamide powder, 5·0 g., was insufflated, a pelvic drain inserted, and the abdomen closed.

PROGRESS.—An intravenous drip of 5 per cent glucose in normal saline was continued after operation, with sulphamezathine 1·0 g. per litre. Penicillin, 100,000 units, was given at once and repeated three-hourly. Progress was excellent and he remained apyrexial throughout. There was a slight leakage of urine from the loin during the first 24 hours. The rectal tube and intraperitoneal drain from the loin were removed on the second day and the other drains on the fourth. An intravenous pyelogram was carried out on the twelfth day, which showed no abnormality and both kidneys were functioning well. The patient was discharged home on the seventeenth day.

Eight months later he was readmitted with acute intestinal obstruction, which on laparotomy was found to be due to a band obstructing the lower ileum. This was divided and he made a good recovery. During convalescence a further intravenous pyelogram was done, and this again showed both kidneys functioning normally.

COMMENT

Gabriel (1945) points out that impalement wounds of the rectum are extremely dangerous. He describes a rather similar case; a girl had been jumping from one chair to another, when a chair collapsed and its broken leg went up her rectum. In this case as well as injury to the vagina there was considerable extraperitoneal extravasation which necessitated a colostomy. In the present case there was no involvement of extraperitoneal tissues; a flatus tube passed beyond the wound in the bowel gave adequate decompression, and with a heavy dosage of penicillin convalescence was uneventful.

Hamilton Bailey (1942) emphasizes that ureteric injuries are exceedingly rare even in military practice and are usually associated with multiple injuries. Gordon-Taylor (1940) states that the ureter may close spontaneously in a few fortunate cases, but that most run the risk of developing a ureteric stricture or a pyelonephritis. There were no signs of urinary infection in the present case and no leakage of urine after the first day. A pyelogram eight months after the injury showed no stricture of the ureter or hydronephrosis.

Finally a search of the literature has not revealed a previously recorded case in which these two comparatively rare injuries have been produced simultaneously.

I am indebted to Mr. J. S. Ramage for his advice and encouragement, and for permission to publish this report.

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GABRIEL, W. B. (1945), *Rectal Surgery*, 3rd ed. London: H. K. Lewis.

GORDON-TAYLOR, G. (1940), *War Wounds and Injuries* (Fletcher and Raven). London: Edward Arnold.

REVIEWS AND NOTICES OF BOOKS

Cancer: Diagnosis, Treatment, and Prognosis. By LAUREN V. ACKERMAN, M.D., and JUAN A. DEL REGATO, M.D., Ellis Fischel State Cancer Hospital. 9¾ × 6⅞ in. Pp. 1115, with 745 illustrations and 9 coloured plates. 1947. London: Henry Kimpton. 100s. net.

IT was bound to come—*the cancer specialist.*

It will be noted that neither author is a surgeon. One is a pathologist and the other a radiotherapist. Such a team cannot function properly as the final court of appeal for the unfortunate patient afflicted with cancer. One cannot suspect the disease in the earlier stages before it is definitely malignant, and the other cannot always treat it adequately and it is the first treatment which is the one that matters. Nor is there a clinician so wise and experienced that he can diagnose the disease in all its many manifestations, nor is there a surgeon who can treat it successfully in every organ in which it may appear. What is the result? The book is just an abstract of various other books. Each organ is dealt with under the headings of Anatomy, Pathology, Clinical Evolution, Diagnosis, Special Examination, Differential Diagnosis, Treatment and Prognosis. A specific example of the summing up which after all determines the treatment can be given (p. 568): "If a small bowel tumour is diagnosed from the clinical history, physical findings and roentgenologic examination, an exploratory laparotomy should be done.

"Before exploration, however, if there has been any degree of obstruction, the electrolyte balance should be restored by appropriate measures, and if there has been any degree of bleeding transfusions should be given.

"At the time of exploration the bowel can be opened and a biopsy made. At times, frozen section can determine whether the tumour is benign or malignant. If the lesion is situated in the terminal ileum and there are large yellow metastases in the liver frozen section usually shows the lesions to be a carcinoid. Even in the presence of liver metastases extensive surgical procedures are justified. If the tumour is malignant or if there is any question of neoplastic change then a radical rather than a conservative operation should be done with removal of the accompanying mesentery and draining lymph-node areas."

Similar examples of what might truly be described at least as very inadequate summing up could be taken from various parts of the book.

This is only to be expected if the cases are not referred to the proper clinicians and surgeons.

The second chapter by Michael B. Shimkin, M.D., consisting of thirty pages is devoted to Cancer Research. Sketchy though it must necessarily be, it is the best part of the book. It does touch on the fringe of most aspects of the subject, and leaves the reader with no clear idea as to the trend of any opinion as to the cause, but states that "Cancer Research must advance on two broad fronts: (1) the study of carcinogenesis, which may eventually lead to the prevention of neoplastic diseases; and (2) the study of tumour growth, which may be the basis of therapy".

This is a very safe and non-committal conclusion and leads really nowhere.

The illustrations are profuse in quantity rather than striking in quality. The coloured illustrations leave much to be desired. The whole book illustrates very adequately that there is no such person as a Cancer Specialist.

The effect of the whole can be summed up, thus: it attempts too much, so achieves too little.

Diagnosis in Daily Practice. By BENJAMIN V. WHITE, M.D., Assistant Clinical Professor of Medicine, Yale University School of Medicine, and CHARLES F. GESCHICKTER, M.D., Professor of Pathology, Georgetown University Medical School. 9¾ × 7 in. Pp. 693 + xvi with 360 illustrations. 1948. Philadelphia and London: J. B. Lippincott Co. £4 10s. net.

IT is difficult to assess the value and usefulness of this large volume, for although it is written for the practitioner it contains much material which would be of more value to the specialist. The authors have attempted to incorporate too much in the book and in consequence too little is written about important conditions. In fact the book is an illustrated dictionary *de luxe.*

It must be acknowledged that the illustrations are excellent, especially the coloured plates. However, the series of charts and tables should prove of more value to the undergraduate than the postgraduate.

Handbook on Fractures. By DUNCAN EVE, jun., M.D., F.A.C.S., Nashville, in collaboration with TRIMBLE SHARBER, A.B., M.D., Nashville. 9 × 6 in. Pp. 263 + xi, with 129 illustrations. 1948. London: Henry Kimpton. 25s. net.

IN this traumatic age the diagnosis and treatment of fractures is of great importance and, although books on fractures are numerous there is always room for good books.

This monograph of two hundred and sixty pages is a concise, readable exposition on the treatment of fractures. The plaster technique, bone-grafting, and other operative procedures are well described and illustrated.

This book covers the subject in the sound practical way that any handbook should, and we can recommend it to students and practitioners alike.

Hernia. Anatomy, Etiology, Symptoms, Diagnosis, Differential Diagnosis, Prognosis, and Treatment. By LEIGH F. WATSON, M.D., F.I.C.S., Los Angeles. Third edition. 9½ × 6¾ in. Pp. 732, with 323 illustrations. 1948. London: Henry Kimpton. 67s. 6d. net.

THE third edition of this well-known monograph has been thoroughly revised and brought up to date with a very modern bibliography at the end of each chapter.

During the second world war one of the great problems the surgeons of this country had to solve was the most efficient operation for the cure of inguinal hernia. Prior to the commencement of hostilities there was a definite trend to perform fascial graft operations for most types of inguinal hernia. However it was soon discovered that fascial grafts were not required for the average recruit and the results on the whole were poor. By the time hostilities were over there was a great swing back to simple excision of the sac where the abdominal musculature was satisfactory. The rehabilitation of the patient was an important part in the after-treatment of these cases.

The American Army had the same conditions to contend with and the surgeons came to the same conclusion as those in this country.

Professor Watson has given the surgeons a very complete book on the anatomy, aetiology, symptoms, diagnosis, differential diagnosis, prognosis, and treatment of hernia. It is the best volume that has so far been published and every medical man who is interested in any aspect of hernia should read it.

The illustrations are uniformly good and the bibliography and references excellent; it is refreshing to see that British literature has been adequately included.

As a reference volume this book could not be bettered.

Congenital Malformations. A Study of Parental Characteristics with Special Reference to the Reproductive Process. By DOUGLAS P. MURPHY, M.D., F.A.C.S., Assistant Professor of Obstetrics and Gynecology, University of Pennsylvania. Second edition. 9 × 6 in. Pp. 127 + viii, with 7 illustrations. 1948. Philadelphia and London: J. B. Lippincott Co. 30s. net.

THE second edition of this book has been considerably enlarged and contains some very practical and useful information. Up to the present time very little has been known of the real background of congenital malformations, but now, owing to the very extensive researches of Dr. Murphy, we are given the facts which do fill in some gaps in our knowledge. His investigations have been very detailed and must have been time consuming.

Some interesting facts emerge from this book. About 25 per cent of congenitally malformed persons are stillborn, and the malformation rate among white persons is about twice the rate found among negroes.

There is no universal frequency of malformations among the illegitimate, which at first sight appears curious.

The older the mother the more likely she is to give birth to a congenitally malformed child.

Periods of relative sterility are very likely to precede the birth of congenitally malformed children.

There is only suggestive evidence to indicate that an attack of maternal rubella, prior to conception, will influence the development of any following offspring.

An Index of Treatment. A Complete Guide to Treatment in a Form convenient for Reference. Edited by Sir ROBERT HUTCHISON, Bart., M.D., LL.D., F.R.C.P., assisted by REGINALD HILTON, M.A., M.D., F.R.C.P. With the Collaboration of 74 Contributors. Thirteenth edition. 9¾ × 6⅝ in. Pp. 972 + xii, with 100 illustrations. 1948. Bristol: John Wright & Sons Ltd. £4 4s. net.

THE new thirteenth edition of this well-known index of treatment has been very thoroughly revised by 74 contributors. The whole of medicine is covered in this comprehensive book on treatment. The editors have been very fortunate in the selection of the numerous contributors, for every branch is dealt with by a specialist in his own particular subject.

As a book of reference this volume could not be bettered, for the specialist, the practitioner, and the student will find a wealth of information within its pages.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., F.A.C.S., F.I.C.S., F.R.S.E., Surgeon and Surgeon-in-charge of the Genito-urinary Department, Royal Northern Hospital, London; etc. Eleventh edition. To be issued in Four Parts. 8¾ × 5⅝ in. Part I. Pp. 100 + xii, with 178 illustrations, a large number in colour. 1948. Bristol: John Wright & Sons Ltd. 8s. 6d. each part.

WE must confess to receiving something of a shock on seeing the eleventh edition of this book clad only in a paper cover; knowing its outstanding merit we felt it worthy of something better. The preface reassures us on this point; the difficulties of production, especially in blockmaking, paper supply, and book-binding, are graphically described and leave the reader in no doubt that the author has followed the right course in bringing out *Part I* in this form in order to make it available in the shortest possible time. There are to be three more parts and we hope that the publisher will then be able to make binding cases available for the whole volume.

The contents reach the high standard of excellence we have come to expect from Mr. Hamilton Bailey. The photographic illustrations, both black and white and coloured, are in many cases superb, and the editorial matter is reduced to the minimum compatible with an adequate description of the subject. All students should possess it, and many a surgeon will have cause to bless the day he added it to his library.

Modern Trends in Diagnostic Radiology. Edited by J. W. MCLAREN, M.A., M.R.C.S., L.R.C.P., D.M.R.E., Director, X-ray (Diagnostic) Department, St. Thomas's Hospital, London; Examiner in Radiology, University of London. 9⅛ × 6⅝ in. Pp. 464 + xxi, with 381 illustrations. 1948. London: Butterworth & Co. (Publishers) Ltd. 60s. net.

WE are becoming familiar with a new series of publications published under the title of "Modern Trends" and so far they have been most useful, practical, and informative volumes.

It is therefore refreshing to see another volume dealing with diagnostic radiology. This book is dedicated to Sir Charles Max Page, who in the past was a strong advocate of the use of X rays in injuries to the skeleton and had an X-ray installation in his own house.

Lord Horder writes a Foreword and is enthusiastic about the great help diagnostic radiology gives to the clinician.

There are some thirty-one chapters in this book and each chapter is written by an expert in each particular branch of diagnostic radiology. Some of the authors come from the United States, France, Denmark, Norway, Iceland, and South Africa, while the majority are attached to X-ray departments in our large teaching hospitals.

Dr. Baclesse, of Paris, contributes an excellent article on Radiographic Diagnosis of Tumours of the Pharynx and Larynx. The radiology of the heart is dealt with by Dr. P. H. Wood, while Dr. Nordentoft, of Copenhagen, writes on colonic disease in children.

Surgeons will be especially interested in the chapters devoted to: Tumours of Bone, Vertebral Disk Lesions, Abnormalities of the Spine, Bone Changes following Irradiation.

Perhaps the most stimulating chapters are those dealing with Ventriculography and Encephalography, by the late E. Lysholm, of Stockholm, and Cerebral Angiography, by Engeset and Kristiansen, of Oslo. These are exceptionally good and beautifully illustrated.

Sir Lionel Whitby contributes a useful chapter on blood changes, which will prove of real value to all who work in X-ray departments.

We have nothing but praise for this book, for it is the last word on this important subject. It should prove of value to the X-ray worker and the clinician and will remain the reference volume on diagnostic radiology for several years to come.

The book is a typical Butterworth production—clear type, excellent reproduction of radiographs, and good paper.

The Clinical Picture of Thyrotoxicosis. By PETER MCEWAN, M.A., M.B., Ch.B., F.R.C.S. (Edin.), Honorary Consulting Surgeon to the Bradford Royal Infirmary. 8¾ × 5½ in. Pp. 127 + xii, with 4 plates and 3 charts. 1948. Edinburgh: Oliver & Boyd Ltd. 15s. net.

THIS small monograph is the outcome of the experience of a general surgeon who is interested in the diseases of the thyroid gland.

The clinical picture of thyrotoxicosis is well portrayed, both in its common form and in its rarer types. It is interesting and refreshing to read the full clinical history of the more difficult cases of thyrotoxicosis and to see how the pre-operative and post-operative treatment plays so important a part in the recovery of the patient.

Although this book is intended primarily to help the practitioner, there can be no doubt that physicians and surgeons will greatly benefit by reading it.

Acute Intestinal Obstruction. By RODNEY SMITH, M.S., F.R.C.S., Assistant Surgeon, St. George's Hospital, London. With a chapter on Radiological Diagnosis by ERIC SAMUEL, M.D., F.R.C.S., F.F.R., D.M.R.E., late Radiologist, The Middlesex Hospital, London. 8½ × 5⅝ in. Pp. 259 + xii, with 102 illustrations. 1948. London: Edward Arnold & Co. 18s. net.

DURING the last ten years great strides have been made in the diagnosis and treatment of acute intestinal obstruction, the real advance being made during the Second World War when suction drainage was put to a severe test and proved its worth in cases of abdominal wounds.

Rodney Smith, who delivered a Hunterian Lecture on Intestinal Obstruction last year, gives an excellent survey of the various varieties of intestinal obstruction and stresses the value of suction drainage not only in cases of obstruction but also as a prophylactic measure. He also insists, quite rightly, on the necessity for control of dehydration, the control of proximal intestinal dilatation, and the avoidance of peritoneal soiling during operation.

This book is full of sound practical surgical common sense and it should be read by all surgeons who have to deal with cases of acute intestinal obstruction.

Dupuytren's Contraction. With Special References to Aetiology and Improved Surgical Treatment, Its Occurrence in Epileptics, Note on Knuckle-pads. By TOND SKOOG, Uppsala. Being Supp. 139, Vol. XCVI, *Acta chirurgica Scandinavica*. Translated by ERICA ODELBERG. 9⅛ × 5⅞ in. Pp. 190, with 50 illustrations. 1948. Stockholm: Norstedt & Söner. No price given.

THIS special monograph of just under two hundred pages is devoted to one surgical affection—namely, Dupuytren's contraction. The author has been fortunate in having a vast amount of clinical material at his disposal for he was able to examine some 69 hands after operation. The material for the most part was seen at the Plastic Surgical Unit of the Queen Victoria Hospital, East Grinstead, where the author was studying as a British Council Scholar under Sir Archibald McIndoe.

Skoog produces conclusive evidence in his cases that hereditary factors play a role in the aetiology of the disease. This does not, however, exclude the possibility that certain environmental factors are of significance.

The writer expresses fresh views on the pathogenesis of the disease and brings forward strong arguments in favour of the view that partial ruptures in the aponeurotic tissue are the origin of the pathological changes.

The treatment advocated is complete removal of the palmar fascia through a transverse incision making use of one of the palmar creases so that scarring is minimal. Quite often an extra Z-shaped incision over the base of the contracted little finger is also required.

This is a very interesting and practical monograph which will always remain as one of the useful books for the modern surgeon.

Textbook of Anæsthetics. By R. J. MINNITT, M.D. (Liverp.), D.A. (R.C.P. & S.), and JOHN GILLIES, *M.C.*, M.B., Ch.B. (Edin.), F.R.C.S. (Edin.), D.A. (Eng.). Seventh edition. 8½ × 5½ in. Pp. 568 + viii, with 229 illustrations. 1948. Edinburgh: E. & S. Livingstone. 30s. net.

THE seventh edition of this excellent text-book contains much new material, and most of the recent developments in anæsthesia and analgesia are included in its pages.

A new chapter entitled Some Legal Aspects is the best we have read upon this interesting subject, and the authors, in conjunction with Mr. J. Crossley Vaines, are to be congratulated upon their achievement in producing such a clear and concise account of legal liability as it affects the anæsthetist. The paragraph on Vicarious Liability for Negligence is most informative, and a summary of the famous Collins *v.* Hertfordshire County Council case (*supra*) provides interesting reading.

There is an excellent new chapter on Curare, and the authors are indebted to Professor John Boyes for an able revision of the subsection on Regional Analgesia in Dentistry.

The publishers deserve a special word of praise for the beautiful production of this useful text-book, which we recommend to undergraduates and also to post-graduates preparing for the Diploma in Anæsthetics.

Recent Advances in Anæsthesia and Analgesia, including Oxygen Therapy. By C. LANGTON HEWER, M.B., B.S., M.R.C.P., D.A., Senior Anæsthetist, St. Bartholomew's Hospital. Sixth edition. 8 × 5¼ in. Pp. 380 + viii, with 149 illustrations. 1948. London: J. & A. Churchill Ltd. 21s. net.

IT is just over four years since the last edition of this book on anæsthesia and analgesia was published and much has happened in this speciality during the interval. The introduction of curare and its extended use in abdominal surgery where relaxation is required has given the anæsthetist a new potent drug.

This book covers every aspect of anæsthesia and the author gives practical advice on the best type of anæsthetic for different kinds of operations. The student and practitioner alike will find much useful information within its pages.

Living Anatomy. A Photographic Atlas of Muscles in Action and Surface Contours. By R. D. Lockhart, M.D., Ch.M., Regius Professor of Anatomy, University of Aberdeen. 9⅞ × 6⅛ in. Pp. 72, containing 149 illustrations. 1948. London: Faber & Faber Ltd. 12s. 6d. net.

THIS book fills in a gap in the modern teaching of anatomy. It is an anatomical study containing 149 photographs of the living subject, male and female.

Professor Lockhart has always been interested in the study of muscular action and this book is the outcome of that study. It should stimulate the student to examine the muscles in action in his own body so that he can obtain a pictorial impression and not one from the dead body.

In this book for the most part the muscles are shown in action by the resistance of movement between the model and the demonstrator or between two models.

To-day, when rehabilitation is so much to the fore, this book will have an added interest for it will prove invaluable to the physiotherapist who wishes to rehabilitate certain muscles in a patient.

An excellent production and a real advance in the teaching of living anatomy.

The Science and Practice of Surgery. By W. H. C. ROMANIS, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), Senior Surgeon and Lecturer on Surgery, St. Thomas's Hospital; and PHILIP H. MITCHINER, C.B., C.B.E., T.D., M.D., M.S. (Lond.), F.R.C.S. (Eng.), D.Ch. (Durham), Hon. Surgeon to H.M. The King; Hunterian Professor of Surgery, Royal College of Surgeons of England; Surgeon and Lecturer in Surgery, St. Thomas's Hospital. In two volumes. Eighth edition. 9¼ × 6 in. Vol. I, General Surgery. Pp. 892 + vi, with 409 illustrations and 20 plates. Vol. II, Regional Surgery. Pp. 955 + viii, with 316 illustrations and 8 plates. 1948. London: J. & A. Churchill Ltd. 1948. Each volume 25s. net.

IN the eighth edition this popular text-book celebrates its majority; it is pleasing to note that in twenty-one years it has increased by only one hundred pages, although there is now no chapter on diseases of the eye. By way of compensation a chapter on plastic surgery has been included. It is a matter for conjecture whether other specialis s might with advantage have been invited to contribute. Although we should not wish to lose the decisive character of the work which has been imprinted on it by the authors, surgery has now become so specialized as to demand a considerable distribution of labour in the writing of a student's text-book.

As an account of the general surgery which the student must know for qualification and even for the higher surgical examinations, the book is excellent. It is eminently readable and as complete as any such work can be. It is adequately illustrated, although the modern student might reasonably expect colour in some of the pictures. It gives a knowledge of surgery based on sound anatomical and pathological principles, and can be recommended without hesitation.

British Surgical Practice. General Editors: Sir ERNEST ROCK CARLING, F.R.C.S., F.R.C.P., Consulting Surgeon, Westminster Hospital, and J. PATERSON ROSS, M.S., F.R.C.S., Surgeon and Director of Surgical Clinical Unit, St. Bartholomew's Hospital; Professor of Surgery, University of London. In eight volumes. 8⅝ × 6⅝ in. Volume III. Pp. 524 + xxxvi, with 37-p. index. 287 illustrations. 1948. London: Butterworth & Co. Ltd. Price per volume £3, Index £1.

THE third volume of this great surgical work contains some sixty-three articles by various specialists. Sir William Gilliatt writes on cæsarean section and Mr. G. W. Black on the surgery of the eyelids. Between these two articles are a series of articles on special branches of surgery, including the ear, eye, diabetes in relation to surgery, endoscopy, bronchoscopy, gastroscopy, peritoneoscopy, urethroscopy, and endometriosis.

Each section is a masterpiece in itself, for the reader is given a lucid, practical discourse on the subject under consideration.

The illustrations are numerous and clear and most of them are original.

Sir Hugh Devine contributes an excellent and comprehensive article on cancer of the colon and is able to draw on his vast clinical knowledge and give the reader a considered opinion concerning the operative mortality of this type of cancer.

Tubbs writes on the surgery of ductus arteriosus and discusses the two methods of approach, the anterior and the lateral, and stresses the point that the latter gives better exposure.

Fat and Air Embolism are ably dealt with by Frazer and Magnus.

The whole volume is a wealth of information and a real credit to the numerous contributors.

Sir Ernest Rock Carling and Professor Paterson Ross are to be congratulated on the able manner in which they have collected such a magnificent team of contributors.

The Surgery of Abdominal Hernia. By GEORGE B. MAIR, M.D., F.R.F.P.S.G., F.R.C.S.E., Surgeon, Law Junction Hospital, Lanarkshire. 8½ × 5½ in. Pp. 408, with 138 illustrations. 1948. London: Edward Arnold & Co. 25s. net.

SURGEONS as a whole are interested in common conditions, especially if these common conditions are difficult to cure. The surgery of abdominal herniæ has a large appeal because of its universal occurrence in every land and the difficulty of obtaining a high percentage of operation cures.

Mr. Mair has contributed a useful practical monograph dealing with this condition; his approach is the right one, namely, from the clinical or practical angle, and he has given his opinion as to the best form of treatment, but one would have liked a little more of his ideas and less of the opinions of the surgeons of the past. Although there is an extensive bibliography at the end of each chapter, yet several of the more important papers on hernia which have been published during the last ten years are not even recorded.

The illustrations are numerous and well drawn and do enhance the value of the text.

The book will take its place as a useful volume on a common condition and should prove of value to both student and practitioner as well as the practical surgeon.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.]

The Treatment of Acute Craniocerebral Injuries due to Missiles. By DONALD D. MATSON, M.D., Research Fellow in Surgery, Harvard Medical School. Being No. 22, *American Lecture Series*. 8½ × 5½ in. Pp. 90 + viii, with 27 illustrations. 1948. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell.) 10s. 6d. net.

Intracranial Tumours. By PERCIVAL BAILEY, Professor of Neurology and Neurological Surgery, University of Illinois. Second edition. 9⅝ × 6½ in. Pp. 478 + xxvi, with 155 illustrations, and 16 plates. 1948. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell.) 55s. net.

Textbook of Genito-urinary Surgery. Edited by H. P. WINSBURY-WHITE, M.B., Ch.B., F.R.C.S. (Eng. and Edin.), Surgeon, St. Paul's Hospital for Genito-urinary Diseases, London; etc. 9⅝ × 6⅞ in. Pp. 1046 + xvi, with 451 illustrations, many in full colour. 1948. Edinburgh: E. & S. Livingstone Ltd. 90s. net.

Modern Surgery for Nurses. Edited by F. WILSON HARLOW, M.B., B.S. (Durham), F.R.C.S. (Eng.), Surgeon, Royal Isle of Wight County Hospital, etc. With a Foreword by Sir LANCELOT E. BARRINGTON-WARD. 8½ × 5½ in. Pp. 795 + xxiv, with 429 illustrations. London: William Heinemann Medical Books Ltd. 25s. net.

Injentos de Piel. By ALBERTO RAUL BEAUX, Professor Libre de Medicina operatoria de la Facultad de Ciencias médicas de la Universidad nacional de la Plata. 10⅜ × 7 in. Pp. 257, with 138 illustrations. 1947. Buenos Aires: El Ateneo. Price not stated.

The Mechanism of Abdominal Pain. By V. J. KINSELLA, M.B., Ch.M. (Sydney), F.R.C.S. (Eng.), F.R.A.C.S., Hon. Surgeon, St. Vincent's Hospital, Sydney. 9¾ × 5⅞ in. Pp. 230 + xiv, with 17 illustrations and 8 plates. 1948. Sydney: Australasian Medical Publishing Co. Ltd. 32s. 6d.

Andreas Vesalius Bruxellensis. The Bloodletting Letter of 1539. An Annotated Translation and Study of the Evolution of Vesalius's Scientific Development. Edited by JOHN B. DE C. M. SAUNDERS, F.R.C.S., and CHARLES DONALD O'MALLEY. 9 × 6 in. Pp. 94. 1948. London: William Heinemann Medical Books Ltd. 21s. net.

Fuss und Bein. Ihre Erkrankungen und deren Behandlung. By Prof. Dr. med. GEORG HOHMANN, Director of Orthopædic Clinic, University, Munich. Fourth edition. 9¾ × 6½ in. Pp. 504 + viii, with 45 illustrations. 1948. Berlin: Springer. DM. 36; bound DM. 39.

The Present Status of the Surgical Treatment of Hyperthyroidism. By WARREN H. COLE, M.D., Professor and Head of the Department of Surgery, University of Illinois College of Medicine, Chicago, Ill., and EDSON FAIRBROTHER FOWLER, M.D., Assistant in Surgery, University of Illinois College of Medicine, Chicago, Ill. Being No. 4, *American Lecture Series*. 8½ × 5½ in. Pp. 81 + viii, with 16 illustrations. 1948. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell.) 10s. 6d. net.

Treatment by Manipulation. By A. G. TIMBRELL FISHER, *M.C.*, M.B., Ch.B., F.R.C.S. (Eng.), Orthopædic Surgeon to the Rheumatic Unit, St. Stephen's Hospital; etc. Being the Fifth Edition of *Manipulative Surgery*. 8½ × 5⅜ in. Pp. 275 + ix, with 126 illustrations. 1948. London: H. K. Lewis & Co. Ltd. 25s. net.

Minor Surgery. By R. J. MCNEILL LOVE, M.S. (Lond.), F.R.C.S. (Eng.), Surgeon, Royal Northern, Mildmay Mission, and Metropolitan Hospitals; etc. Third edition. 7¼ × 4⅞ in. Pp. 430 + viii, with 221 illustrations. 1948. London: H. K. Lewis & Co. Ltd. 22s. 6d. net.

Identification of Tumours. Essential Gross and Microscopic Pathologic Features systematically arranged for Easier Identification. By N. CHANDLER FOOT, M.D., Professor of Surgical Pathology, Cornell University Medical College; etc. 9 × 6 in. Pp. 397 + xx, with 241 illustrations. 1948. Philadelphia and London: J. B. Lippincott Co. 36s. net.

The Digestive Tract in Roentgenology. By JACOB BUCKSTEIN, M.D., Assistant Professor of Clinical Medicine, Cornell University Medical College; etc. 10 × 7 in. Pp. 889 + xvi, with 659 illustrations. 1948. Philadelphia and London: J. B. Lippincott Co. £5 net.

Nouveau Précis de Pathologie chirurgicale. By J. PATEL (Editorial Secretary), J.-P. CALVET, J. CAUCHOIX, R. COUVELAIRE, J. GOSSET, J. HEPP, L. LEGER, P. MALLET-GUY, J. MATHEY, R. MERLE D'AUBIGNÉ, A. MOUCHET, P. PETIT, V. VEAU, M. ARNAUD, H. BONNET, and J. DELARNE. To be issued in six volumes. 8⅜ × 6½ in. Vol. I, Pathologie général; Pathologie tissulaire générale; Affections des Membres et des Ceintures. Pp. 687 + viii, with 309 illustrations. Vol. II, Affections des Membres et des Ceintures (suite). Pp. 620 + viii, with 356 illustrations. 1947. Paris: Masson et Cie. Vol. I, 1500 fr.; Vol. II, 1300 fr.

The Radiology of Bones and Joints. By JAMES F. BRAILSFORD, M.D., Ph.D., F.R.C.P., F.I.C.S., Director of Radiological Studies in Living Anatomy, The University of Birmingham; etc. Fourth edition. 9⅝ × 7¼ in. Pp. 760 + xvi, with 615 illustrations. 1948. London: J. & A. Churchill Ltd. 63s. net.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., F.R.S.E., Surgeon and Surgeon-in-Charge of the Genito-urinary Department, Royal Northern Hospital, London; etc. Eleventh edition. To be issued in Four Parts. 8¾ × 5⅝ in. Part II. Pp. 101–196 + iv, with 182 illustrations, a large number in colour. 1948. Bristol: John Wright & Sons Ltd. 8s. 6d. each part.

An Introduction to Surgery. By RUTHERFORD MORISON, M.D., F.R.C.S., M.A., D.C.L., LL.D. and CHARLES F. M. SAINT, C.B.E., M.D., M.S., F.R.C.S., F.R.A.C.S. Fourth edition. 8⅝ × 5⅝ in. Pp. 330 + xii, with 304 illustrations. 1948. Bristol: John Wright & Sons Ltd. 42s. net.

Hæmostatic Agents. By WALTER H. SEEGERS, M.S., Ph.D., Professor of Physiology, Wayne University College of Medicine, Detroit, Mich. and ELWOOD A. SHARP, M.D., Sc.D., Director, Department of Clinical Investigation, Parke Davis & Co.; Lecturer, Department of Medicine, Wayne University College of Medicine, Detroit, Mich. 9 × 6 in. Pp. 131 + xii, with 27 illustrations. 1948. Springfield, Ill.: C. C. Thomas. (Oxford: Blackwell.) 25s. net.

The Treatment of Acute Compound Injuries of the Spinal Cord due to Missiles. By DONALD D. MATSON, M.D. Being No. 23, *American Lecture Series*. 8½ × 5½ in. Pp. 64 + viii, with 12 illustrations. 1948. Springfield, Ill.: C. C. Thomas. (Oxford: Blackwell.) 9s. 6d. net.